

Appendix I

Exposure Parameter Variability Analysis

DRAFT MEMORANDUM

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SUBJECT: Exposure Factor Variability Analysis (Options 1 and 2)

I. Introduction

This memorandum describes the methodology used in the Hazardous Waste Combustor (HWC) risk analysis to evaluate the impact of exposure parameter variability on risk estimates for three key receptor population/constituent combinations, including the recreational fisher for mercury and the beef farmer and dairy farmer for dioxin. It also describes the methodology used to evaluate uncertainty introduced into the variability analysis (i.e. uncertainty in identifying the risk associated with specific percentiles of the inter-subject variability distribution describing the variation of risk in the exposed population) through the selection of a subset of facilities and through the finite number of individuals sampled in the vicinity of each facility during Monte Carlo analysis. The problem may be stated as follows: *Considering variability of exposure factors across individuals in the exposed population, what is the risk (probability of cancer or hazard quotient) associated with an individual at the Xth percentile (e.g. 95th percentile) of the cumulative frequency distribution in the exposed population, and what is the confidence interval surrounding this estimate?*

The HWC methodology utilizes a post-processing approach wherein exposure parameter variability is incorporated into the cumulative distribution of risk in the exposed population after that distribution has been generated using only central tendency values for exposure factors. As a baseline, calculations of risk were performed using only mean values for all exposure factors. This distribution of risk in the exposed population represents the best estimate of risks to individuals when only variability of exposure conditions (and not exposure factors) is considered. In effect, it represents the distribution of risk in the population if all individuals in a geographic sector have the mean exposure factors for individuals in that sector. After generating this baseline distribution of risks, the methodology replaced all mean values of the exposure factors by an inter-subject variability distribution. The means for these inter-subject variability distributions were selected to be the same as the mean values used in the baseline assessment. The process of generating the inter-subject variability for risk (probability of cancer or hazard quotient) then was repeated and a new variability distribution generated that reflected both variability of exposure and variability of exposure factors. As will be described below, this post-processing approach was accomplished in three steps:

- # The baseline variability analysis, incorporating variability of exposure but not variability of exposure factors, was produced. This distribution was stored for later comparison with the full, post-processing, variability analysis. The same analysis produced the mean estimate of risk in each geographic sector surrounding a facility. The population-weighted distribution of these means across the different sectors constitutes the probability density function produced in the baseline analysis.

- # The risk (probability of cancer or hazard quotient) for each geographic sector generated by the baseline analysis was retrieved. The inter-subject variability in the risk associated with a given sector then was determined by using the same exposure conditions as in the baseline analysis for that sector, but allowing inter-subject variability of exposure factors (i.e. probability density functions for each exposure factor). The mean values of these probability density functions were equal to the point estimate value used for that exposure factor in the baseline assessment. This step produced one probability density function, reflecting inter-subject variability of risk, for each geographic sector.
- # The resulting probability density functions from the previous steps (one for each geographic sector) were combined through population-weighting to obtain the new inter-subject variability distribution for risk in the entire exposed population. This new distribution reflects variability in both exposure and exposure factors.

The HWC post-processing methodology above is designed to handle receptor populations whose risk is dominated by a single exposure pathway (e.g., in the case of the beef farmer, risk for dioxin is dominated by beef ingestion). The presence of a single risk-driving pathway for the three key receptor population/constituent combinations evaluated in the HWC variability analysis simplifies the methodology needed to assess the impact of exposure parameter variability, since it is not necessary to evaluate multiple exposure pathways separately for each receptor population. If there is a single pathway, and if the same inter-subject variability distributions for exposure factors apply to all geographic sectors (as was assumed here), it is possible to apply the post-processing approach directly to the risk estimate for each geographic sector as described above. If multiple pathways contributed significantly, it would be necessary to decompose the risk in each geographic sector into the contributions from each pathway, apply the variability of exposure factors to each pathway, and then recombine these new variability distributions within the sector. As described later, a single pathway did dominate in each sector (contributing more than 95 of the risk), and was the same in each sector (for a given constituent), and so the post-processing approach used here is valid.

The resulting HWC methodology evaluates the aggregate impact of inter-subject variability associated with three exposure factors: (1) ingestion rate per unit body mass, (2) occupancy period, and (3) age crossing correction factor (note: the latter two factors do not apply to the recreational fisher for which noncarcinogenic hazard quotients [HQs] are generated). *This memorandum describes the rationale and data sources used in deriving the inter-subject variability distributions for these three exposure factors, as well as the uncertainty associated with specific percentiles in the overall variability distribution of risk in the exposed population as a result of sampling issues.* The HWC variability analysis methodology is consistent with guidelines for variability analysis set out in EPA's *Guiding Principles for Monte Carlo Analysis* (EPA, 1997). The uncertainty analysis also is consistent with these guidelines for the components of uncertainty considered here, although this uncertainty analysis did not examine uncertainty introduced by model errors and uncertainty in exposure factors (focusing instead on uncertainties introduced by sampling of facilities to represent source categories and by sampling of a finite number of individuals from the exposure factor inter-subject variability distributions).

In performing the variability and uncertainty analyses, two methodologies were used. The first, Monte Carlo sampling, is the most commonly employed method in risk analysis for treatment of both variability and uncertainty. So long as the underlying distributions (probability density functions) for all parameter values in a model can be specified, Monte Carlo analysis allows analysis of the propagation of variability and uncertainty through the model. The present analysis, however, had a component of uncertainty which is not handled readily by Monte Carlo analysis: some facilities included in a facility category were selected at random from a defined set, and others were selected to be representative of higher-end

exposures from a different defined set. This problem can be addressed through use of the SUDAAN software, which allows determination of uncertainty introduced by non-random sampling. The same software can be used, with modifications discussed later, to determine both the percentiles of the variability distribution for risk in the exposed population, and the confidence intervals around any specific percentile.

Since these modifications to SUDAAN had not been employed as frequently in regulatory analyses as have the Monte Carlo methods, it was decided that the SUDAAN variability results should be benchmarked against a Monte Carlo analysis for the variability results in a simulation where Monte Carlo analyses have been established as appropriate and accurate. This procedure will be described later. The result is that a Monte Carlo analysis of the variability distributions for risk (probability of cancer or HQ) was generated; the modified SUDAAN analysis for these same distributions was generated; and the SUDAAN and Monte Carlo results for identical percentiles in the variability distributions were compared. If the modified SUDAAN approach is valid, specific percentiles of the inter-subject variability distribution for risk should be approximately the same as that produced by the Monte Carlo analysis. A subsequent analysis of these results indicated that the modified SUDAAN procedure produced percentiles of the variability distributions which were essentially the same as those generated under the Monte Carlo procedure, establishing the utility of the modified SUDAAN procedure in generating both the variability distributions and the confidence intervals around the percentiles in those variability distributions.

The following discussion describes the benchmark Monte Carlo analysis of variability first. It then turns to the formulation of both variability and uncertainty within the SUDAAN analysis. It closes with a discussion of the comparison between the Monte Carlo and SUDAAN results, establishing the validity of the modified SUDAAN procedure.

II. Problem Formulation for Variability

The HWC risk assessment focuses on the risk of cancer and non-cancer endpoints resulting from exposure of the U.S. population to air emissions from a variety of facility categories. The risk metric for cancer was the lifetime excess probability of cancer, and the risk metric for non-cancer was the hazard quotient. To perform the calculations of risk imposed by a facility category, a representative sample of sites was selected for that category (uncertainty introduced by the fact that this sample was not truly random will be discussed later). For each of these sites, source terms were estimated either through emissions data from that site or by imputation based on data from similar sites. The region surrounding each site was divided into 16 geographical sectors and dispersion calculations performed to estimate the concentration of each pollutant in the environmental media of each sector. Exposures to individuals in a sector then were estimated based on rates of ingestion and inhalation per unit body mass (to obtain average daily rate of intake), as well as exposure duration and loss through food preparation. Finally, these exposures were converted to estimates of risk using measures of sensitivity (slope factors for cancer risk and RfDs/RfCs for noncancer risk). The result is an estimate of the lifetime excess probability of cancer and/or hazard quotient for each individual and each constituent.

In the baseline assessment, “central tendency” calculations of risk were performed using only the mean value for each exposure factor (i.e. the point estimate representing the mean of any inter-subject variability distribution for that exposure factor). This resulted in risk estimates that were the same for all individuals in a given receptor population for a given sector, although there was variation of the risk across sectors due to inter-sector variability of source terms and dispersion factors. From this information, distributions displaying the fraction of exposed individuals with a risk at or below any particular value were developed through population-weighting of the mean risk estimates in the various

geographic sectors. Such an assessment does not fully reflect variability of risk in the exposed population, however, since it does not incorporate inter-individual variability of risk within a sector.

One strategy for considering variability more fully in decision making is to repeat the calculations of risk using inhalation rates, ingestion rates, exposure duration, and food loss factors representative of individuals at the “upper tail” of exposure in a sector (a “maximally exposed” individual). The problem with such an approach is that the selection of multiple exposure factors, each representative of maximally exposed individuals, can lead to the creation of a composite individual with exposure characteristics at the extreme tail of the distribution of risk in a sector. This kind of analysis does not provide full information on the percentile of risk represented by that individual, so it is impossible to state the degree to which that individual is realistic and the fraction of the actual population whose risk will fall above that maximally exposed individual.

To produce a more complete characterization of variability of risk within the exposed population, this project used a formal procedure of Monte Carlo analysis to generate distributions of risk within a sector, and then to “fold together” the variability distributions from separate sectors (through population-weighting) and facilities to produce a composite variability distribution for risk in the entire population exposed to emissions from a facility category. This procedure is consistent with guidelines for variability analysis set out in EPA’s *Guiding Principles for Monte Carlo Analysis* (EPA, 1997), and allows the determination of the fraction of individuals with a risk at or below any particular value of interest (the Monte Carlo analysis did not include an assessment of uncertainty, which will be discussed in the section on the SUDAAN analysis). These individual risks vary across the exposed population due to variability in several of the factors used in the calculations:

- # Inter-facility variability of the source terms for facilities within a category
- # Inter-facility and inter-sector variability in the dispersion factors
- # Inter-subject variability in the exposure factors for a given receptor population;
- # Inter-subject variability in the sensitivity to carcinogens and noncarcinogens in the different receptor populations.

The numerical values of all parameters above are variable in one or more senses; i.e., they vary between facilities, between sectors around a facility, between receptor populations, and/or between individuals in a receptor population within a sector. The study did reflect variability of the source term and dispersion factor within a sector; instead, the exposure conditions were averaged across the geographic region encompassing a sector and this mean exposure condition used for all individuals in that sector. There is, however, variability of the source term between facilities and of the dispersion factor between sectors associated with a facility. ***This variability (of source terms and dispersion factors) is incorporated into the present analysis by using site-specific source terms and spatially-varying dispersion characteristics. The exposure factors, however, are variable across an exposed population even within a single geographic sector, and were represented in the post-processing analysis by a distribution of these factors across the exposed population (with different distributions for different receptor populations in a sector). The variability of sensitivity of individuals to carcinogens and noncarcinogens cannot be characterized at present and cannot, therefore, be considered formally. Instead, it should be noted that these sensitivities were represented here by use of the cancer slope factors and RfDs/RfCs generated under conservative assumptions used commonly as default science policy assumptions. These measures of sensitivity include within them uncertainty factors, modifying***

factors, etc, that are likely to make them representative of upper bound estimates of the values for the most sensitive members of the exposed population.

For any given assessment of risk associated with a facility category, there are N facilities. Associated with each facility is a source term S_{imn} , where the subscript i indicates the facility, the subscript m indicates the environmental medium into which the constituent (compound) is released, and the subscript n indicates the constituent. There are geographic sectors surrounding each facility and the dispersion coefficient (concentration in the environmental medium per unit source term) is DC_{ijmn} , where the subscript i refers to a facility, subscript j refers to a sector associated with that facility, and m and n are as defined previously. For an individual in a sector, assigned to a receptor population, there is an exposure factor EF_{ijmn} which converts concentration in the environmental medium to the average daily rate of intake (ADRI) into the body. For this individual, there is a sensitivity factor SF_{ijmn} which converts from the average daily rate of intake to the probability of cancer or the hazard quotient. In addition, there is a population of size P_{ij} in that sector and specific to that receptor category. This results in two equations, the first for carcinogens and the second for noncarcinogens:

$$Prob_{ij} = \sum \sum S_{imn} \times DC_{ijmn} \times EF_{ijmn} \times SF_{ijmn} \quad (1)$$

$$HI_{ij} = \sum \sum S_{imn} \times DC_{ijmn} \times EF_{ijmn} \times SF_{ijmn} \quad (2)$$

where the first (external) summation is over all constituents to which an individual is exposed and the second (internal) summation is over all exposure pathways. In Equation 1, the lifetime excess probability of cancer is shown as Prob. In Equation 2, HI is the hazard index, or sum over all hazard quotients from constituents acting by a common mechanism of action. In this analysis, there was no summation over constituents (they do not act by a common mechanism of action) and so HI may be replaced by HQ for each constituent and the external summation dropped from Equation 2.

In Equations 1 and 2, there are three sources of variability as discussed previously (sensitivity not being treated as variable). The variability in S_{imn} is treated through making this numerical value site-specific for the different facilities. No further consideration of variability of the source term (e.g. temporal variability) is included in the variability analysis. The variability in DC_{ijmn} is treated through making this numerical value specific to the individual sectors associated with individual facilities. No further consideration of variability of the dispersion coefficient (e.g. temporal variability or variability within a geographic sector) is included in the variability analysis. The dispersion coefficient for the center of a sector is used for all individuals in that sector. Variability of EF_{ijmn} , however, is not accounted for by inter-facility and inter-sector differences in environmental concentrations. To fully characterize variability of the risk in the exposed population, it is necessary to consider inter-subject variability of the exposure factors. With variability of exposure factors incorporated into the assessment, the result is a probability density function describing the variability of risk for cancer, and a probability density function describing the variability of hazard quotient in the exposed U.S. population. Note that in these distributions, the term “exposed” means the population located in one of the sectors associated with facilities contained in a facility category (i.e., it does not mean the total U.S. population).

The variability of exposure factors occurs due to three sources. Individuals differ in these factors as they age; they differ in these factors depending on the kinds of activities in which they engage (e.g., fishing); and there is inter-individual variability even within an age group and activity. The inter-subject variability distributions of risk will differ for these different combinations of age and activity. The present analysis reflects this by developing separate variability distributions for different age/activity populations (referred to here as *receptor populations*). The problem of variability analysis addressed in this report is:

What is the variability of risk (i.e., distributions $Prob_{var}$ and HQ_{var}) in the exposed populations when inter-facility variability of source term, inter-sector variability of dispersion, and inter-subject variability of the exposure factors (EF) are incorporated? Specifically, what are these distributions for different receptor subpopulations defined by age and activity?

For the analysis of hazard quotients associated with recreational fishers (one of the activity categories discussed later), a modified approach to variability estimation and characterization was used. For this category of receptors, there is insufficient information on which to reliably determine the number of recreational fishers in the different age groups for the separate sectors. This precludes developing a population-weighted distribution, HQ_{var} . As an alternative to estimating variability of HQ in the entire exposed population for a facility category, therefore, the present analysis examined only a related but simpler issue for fishing exposures. The question asked was:

For which facilities may it be stated that some specified fraction of the recreational fishers exposed in one or more of the sectors surrounding that facility have a hazard quotient greater than or equal to 1.0? If there were equal numbers of recreational fishers in each sector, what would be the inter-subject variability of HQ across all sectors for the exposed population?

III. Sensitivity Analysis

For the facility categories examined here, a baseline central tendency analysis first was performed to determine the relative importance of different constituents and exposure pathways in Equations 1 and 2. Three receptor populations showed the largest risks for the facility categories examined here (i.e. were the subpopulations whose risks were maximal in the entire exposed population), and were determined to be the “driving” receptor categories for regulatory decisions:

- # Beef cattle farmers
- # Dairy cattle farmers
- # Recreational fishers.

When the separate contributions of the pathways and constituents were examined for these receptor populations, two constituents contributed more than 95% of the total risk in all geographic sectors; these were determined to be the “driving” constituents:

- # Dioxin
- # Methylmercury.

To ensure consideration of all age groups, including young ages, four separate age groups were examined; these were selected on the basis of the relative uniformity of exposure factors across the ages in an age group, with distinct differences in these factors between age groups:

- # 0-5 years
- # 6-11 years
- # 12-19 years
- # 20+ years.

Finally, exposure pathways were considered for these different combinations of receptor population, constituent and age group. In all facility categories and geographic sectors considered in the baseline analysis, the following exposure pathways contributed more than 95% of the risk for all age groups:

- # Ingestion of dioxin in beef for the beef farmer
- # Ingestion of dioxin in milk for the dairy farmer
- # Ingestion of methylmercury in fish for the recreational fisher.

These three exposure scenarios, for all four age groups, therefore represent adequately the risks that will drive regulatory decisions. Inter-subject variability distributions were developed for each. *As is shown in Section IV, inter-individual variability within a receptor population is controlled by several separate factors which appear in multiplicative fashion within the exposure factor. As a result of this multiplication, the exposure factor is equally sensitive to unit changes in each of these components, so sensitivity analysis indicates that each must be considered equally in generating variability distributions.*

IV. Selection of Parameters for Evaluation

The discussion above of the sensitivity analysis for contributions to total risk indicates that for three receptor groups there is a single constituent and exposure pathway which contributes more than 95% of the total risk to individuals in the exposed population. For these cases, the variability distributions for total risk are approximated well by the variability distributions constructed from these dominant constituents and pathways. In other words, including inter-subject variability of exposure pathways and constituents other than those mentioned above would not significantly affect the final variability distribution of risks across the exposed population.

As outlined in Section II, the baseline risk estimates already incorporate inter-sector variability in S_i and DC_{ij} as used in Equations 3 and 4. Variability of EF_{ij} for each of these three constituent/receptor population combinations must, therefore, be specified.

The exposure factor, EF, converts the concentration in an environmental medium (determined by the product of S and DC) to the average daily rate of intake. If C is the concentration in the environmental medium, the average daily rate of intake (ADRI) may be found by:

$$ADRI = C \times IR \times ED / (AT \times BW) \quad (3)$$

where IR is the intake rate of the environmental medium, ED is the exposure duration, AT is the averaging time (taken to be 70 years for carcinogens and equal to ED for non-carcinogens), and BW is the body weight or mass. For carcinogens, the lifetime risk is the product of the ADRI and the slope factor; for noncarcinogens, the value of HQ is equal to the ratio of the ADRI over the RfD or RfC. The exposure factor, EF, may be found by dividing the right-hand side of Equation 3 by C, or:

$$EF = (IR/BW) \times (ED) / (AT) \quad (4)$$

The first term in brackets on the right hand side is the intake rate per unit body mass, and the second is the length of exposure (which depends on the occupancy period). Both are components of the exposure factor considered in this analysis. The averaging time, AT, is a matter of policy. In Equations 3 and 4, ED and AT are not needed for non-cancer effects and so are not used in the calculation of the hazard quotient.

This variability analysis examines three primary components of the value of EF for which there is significant inter-subject variability:

- # Variability in the ingestion rate per unit body mass for beef, milk, and fish (i.e., for both dioxin and mercury exposures). This is the ratio of IR over BW as shown in Equation 4. It is required for both cancer and non-cancer risks.
- # Variability in exposure duration for the beef and milk ingestion (i.e., for dioxin where cancer risks are dominant) but not for fish ingestion (i.e., for mercury where non-cancer risks are dominant and, therefore, ED and AT are not required). This is the value of ED as shown in Equation 4.
- # Variability in a correction factor for crossing age groups. This factor accounts for the fact that the baseline central tendency risk estimates assumed values of IR/BW were constant for an individual beginning exposure in a given age group, whereas the actual aging of an individual may cause movement between age groups during the exposure duration. For example, if an individual began exposure in the 0-5 year age group, and the exposure continued for 7 years, the values of IR/BW used in the baseline calculations were those of the 0-5 age group for all 7 years of exposure, despite the fact that two of those years were spent in the 6-11 year age group. This factor is relevant for the carcinogen exposures (dioxin) but not for the non-carcinogen exposures (mercury) since in the latter case cumulative exposure is not relevant.

Note that there is no variability in the averaging time, AT, in Equation 4 since this is a matter of the definition of the ADRI.

V. Establishment of Exposure Parameter Distributions

These two sources of variability in Equation 4 (IR/BW and ED) as well as the age correction factor were analyzed separately using available data sets. They then were combined for a composite variability distribution for EF. Separate discussions are provided for each of the exposure parameters described above. In each case, lognormal distributions were selected based on the fact that this distributional form often is found for environmental and biological parameters (Morgan and Henrion, 1990; Crawford-Brown, 1996), provided a good fit to the relevant data contained in the *Exposure Factors Handbook*, and compared well to other distributional forms, as discussed later. The lognormal distribution is characterized by a median (M, 50th percentile value) and a geometric standard deviation (GSD). The defining probability density function is:

$$\text{PDF}(x) = \exp\{-(\ln x - \ln M)^2 / (2 \ln^2 \text{GSD})\} / \{2\pi(x \ln(\text{GSD}))\} \quad (5)$$

where exp is the exponential function and pi is 3.1417.

In such a distribution, 68% of the values are contained in the interval defined by the median divided by the GSD and by the median times the GSD. The 95% confidence interval is defined at the lower end by the median divided by the square of the GSD, and at the upper end by the median times the square of the GSD. The GSD, in turn, equals the ratio of the 84th percentile of the distribution divided by the 50th percentile (see the discussion in Crawford-Brown, 1996), or the 50th percentile divided by the 16th percentile (note that the interval from the 16th to 84th percentiles contains 68% of the values of the distribution).

This report does not present a discussion of the particular data sets used to obtain these individual variability distributions. The justification for using these data sets as a basis for estimating exposure to the receptor populations examined here has been summarized previously in the *Exposure Factors Handbook*, and the reader is referred to that reference for a full discussion of the validity of those data

sets. What is noted here is that the data sets described below are specific to the receptor populations to which they have been applied in this analysis, with a difficulty in applying the recreational fisher data as described later.

On the issue of selection of distributional forms, a comparison was made of the best fitting curves from each of several distributions: the lognormal, the gamma, the Weibull and the generalized gamma. In fitting such distributions, several considerations must be taken into account. First, there is the need to select a distribution that succeeds in approximating the data at the upper percentiles (i.e., above the 90th percentile). Second is the need for a good statistical measure (goodness of fit) for the ability of the mathematical form of the distribution function to fit the data. Given the assumption in such goodness of fit measures of a random sample, rather than a survey sample (the latter being the kind of sample underlying the data examined here), a third consideration is that a visual inspection of the best fitting curve for a distributional form should show the fit to be reasonable. This should be backed by inspection of the table of values comparing the data and the best fitting distributional model at specific percentiles. Finally, any choice between marginally different distributions (i.e., distributions with marginally different goodness of fit measures) should reflect the sensitivity of final risk estimates to the inability to select reasonably between these distributions.

As a formal goodness of fit test, the data sets discussed below were examined under Chi Square and p-value tests using the gamma, lognormal, Weibull, and generalized gamma models. In addition, a simpler square of the residuals examination was performed for the lognormal, beta, and normal distributions. For the residuals test, the lognormal distribution provided the smallest residual for all of the parameters fit; this test, however, is the least sensitive. In considering the more rigorous p-value test (the results of which are summarized in Chapter 6 of the Background Document), the lognormal distribution provided either the best, or an approximately equivalent, p value for the majority of the data sets considered (see Table 6-18 of the Background Document). This was particularly true for the beef ingestion parameter values (3 age groups considered), the fish ingestion parameter values (all distributions produced p values below 0.1), and the ACF factor. For beef ingestion, ages 6-11, the p values for the gamma, lognormal, Weibull, and generalized gamma were 0.17, 0.53, 0.091 and 0.314, respectively. For the 12-19 age group, these were 0.57, 0.45, 0.46 and 0.42, respectively. For the adult, these were 0.001 for all models. For the ACF factor, these were 0.2, 0.26, 0.043, and 0.13, respectively.

For the milk ingestion rate and farm occupancy factors, the gamma or generalized gamma provided the better overall fit, although in the case of the occupancy factors all distributions had p values below 0.1. For milk consumption, the p values for the four distributions were 0.4, 0.1, 0.4, and 0.56, respectively, although visual fits to the data were not distinguishable. These two parameter values were determined through surveys of small populations, precisely the form of data least suited to formal p value tests.

To consider the issue of whether the marginally better fits provided by the gamma and generalized gamma distributions, relative to the lognormal distribution, indicated a need to switch to these for at least the milk ingestion rate, a sensitivity analysis was performed considering the first three distributions (the gamma, lognormal, and Weibull; the generalized gamma can be simulated in the Monte Carlo software used here but only with significant effort that was not justified by its marginal improvement over the gamma distribution). The sensitivity analysis was conducted for the product of the milk ingestion rate and farm occupancy factor, the two parameters for which there was even an issue about switching from the lognormal. The analysis simulated a four-sector geographic region. The distributions for the milk ingestion rate and occupancy period were taken from the best fits of the three distributions above to the data contained in the *Exposure Factors Handbook* (where the numerical values corresponding to specific percentiles of the data were provided). The medians for the milk ingestion rate then were adjusted for the four sectors to reflect inter-sector variability of concentrations in milk. One sector was multiplied by 0.1,

one by 0.5, one by 1.0, and one by 5.0. Equal populations were placed into each of the four sectors. A random sample of 5,000 individuals then was drawn using the Monte Carlo (CrystalBall)® methods described elsewhere in this report. The 95th, 97th and 99th percentiles of the resulting population distribution were obtained, and the procedure repeated over the three candidate distributions.

The ratio of the percentile value obtained from a given distributional form over the percentile value from the lognormal form was calculated. For example, the inter-subject variability distribution for the total milk ingestion during the exposure interval under the assumption of a lognormal distribution was generated. Then the 95th, 97th and 99th percentiles of this distribution were obtained. This process was repeated for the gamma distribution, and the same percentiles determined. Then the ratio of the 95th percentile for the gamma distribution over the 95th percentile for the lognormal distribution was calculated (seen to be 0.85 below). The same procedure was done for other percentiles, and for the Weibull distribution. This necessarily produces a ratio of 1.0 for the lognormal distribution itself since that is the reference distribution. For the 3 percentiles and three candidate distributional forms, the following ratios were determined:

95th: 0.85 for the gamma; 1.00 for the lognormal; 1.16 for the Weibull
97th: 0.90 for the gamma; 1.00 for the lognormal; 1.22 for the Weibull
99th: 0.83 for the gamma; 1.00 for the lognormal; 1.25 for the Weibull

It may be noted from this analysis that the sensitivity of the upper percentiles in an aggregated population (aggregated across sectors) is not large when at least four sectors are present. The variations of the ratios from 1.0 will be less when there are more than 4 sectors due to convergence to the mean. There are significantly more than four sectors in the analyses developed for this study, so it is unlikely that the selection of a distribution other than the lognormal for the factors where lognormal did not provide the best p value will be significant. **When this sensitivity analysis is coupled with the good visual fit of the lognormal distribution (an equally valid method of selecting fits for data of this quality), with the fact that the lognormal provided the best, or an equally good, p value in 5 of the 7 parameters considered, and with the ability of the lognormal to retain lognormal properties when one takes the product of lognormally distributed parameters, it was determined that the lognormal distribution would be used throughout this analysis.**

On the issue of correlation, it should be noted that the three factors considered in this variability analysis are statistically independent (i.e., there is no correlation). While ingestion rate (IR) and body mass (BW) are correlated, this analysis uses a data set in which IR and BW were determined for each individual in the population, and the ratio (IR/BW) calculated based on the values specific to that individual. There was no selection of random samples of IR and of BW, so any correlation between IR and BW already is included in the data set taken from the *Exposure Factors Handbook*. The exposure duration (ED) is correlated with the ratio IR/BW, since both quantities are functions of age. Within a subpopulation of a given age, (which is the type of subpopulation used in the present analysis), however, there is no reason to suspect a correlation between ED and the ratio IR/BW, since there is no reason why exposure duration should be related to either intake rate or body mass. Still, this lack of correlation has not been examined empirically to date, so remains a source of uncertainty. The age correction factor also is assumed to be uncorrelated with IR, BW or ED, using the argument advanced above for correlation between IR/BW and ED. Again, this lack of correlation has not been tested to date.

Goodness of fit between the lognormal distributions and the data in the *Exposure Factors Handbook* also was assessed by direct comparison of predictions from the lognormal distribution against specific cumulative percentiles provided in the *Handbook*. These comparisons are provided in Tables 6-20 through 6-23 in the Background Document for this analysis. Agreement at the upper percentiles

generally was to within 20%. In addition, the mean from the lognormal fits was compared against the mean from the data by calculating the ratio of the former over the latter. These ratios varied between 0.92 and 1.17, indicating good agreement on mean values.

In the following discussion, a slight revision to the methodology in Equations 1 and 2 was used. The baseline central tendency risk estimates for each sector first were calculated based on mean values for exposure factors. The composite variability distribution for the exposure factor (IR/BW times ED times ACF for the cancer risks; IR/BW for the non-cancer risks) then was calculated from the following relationships that apply to the product of lognormal distributions:

$$\text{Median of Products} = M_1 \times M_2 \times M_3 \dots \quad (6)$$

where M_i is the median of the i th exposure factor. In addition, the GSD for the product is:

$$\text{GSD}_{\text{product}} = \exp(\ln^2(\text{GSD}_1) + \ln^2(\text{GSD}_2) + \dots + \ln^2(\text{GSD}_n))^{0.5} \quad (7)$$

where GSD_i is the GSD of the i th exposure factor.

The relationship between the mean and median for a lognormal distribution is

$$\text{Median} = \text{Mean} \times \exp(-\ln^2 \text{GSD}/2) \quad (8)$$

where the GSD is for the composite exposure factor distribution as defined in Equation 7. The GSD for the composite exposure factor distribution is known and is the same for all geographic sectors (although varying between the cancer and non-cancer effects due to differences in the number of factors considered in the composite exposure factor). The risk for each sector determined from the baseline analysis (which represented then mean) then was converted to a median value using Equation 8.

Finally, the variability distributions for the exposure factors were normalized by dividing all values in the distribution by the median value. This yields the distribution of the ratios of the exposure factor over the median exposure factor. The resulting variability distribution for EF then is a **normalized distribution** with a median value of 1.0 and the GSD defined previously. This normalized distribution is obtained from the following equation (analogous to Equation 4 with the ACF added):

$$\text{EF}_{\text{ratio}} = (\text{IR/BW})_{\text{ratio}} \times \text{ED}_{\text{ratio}} \times \text{ACF}_{\text{ratio}} \quad (9)$$

where ED and ACF are dropped for the case of non-cancer effects. The distribution of EF_{ratio} then is the distribution of ratios of individual-specific values of EF over the median value for that subpopulation of receptors. The variability distribution of risks in a sector then may be obtained by multiplying this normalized distribution by the median risk for that sector as obtained previously. ***This approach is mathematically identical to one in which the separate, non-normalized, distributions for IR/BW, ED and ACF are used in Equations 1 or 2, but greatly simplifies calculation efforts since the same normalized distribution may be applied to each sector for a given constituent and pathway. In other words, this approach requires that only one normalized distribution be developed for each constituent/receptor population combination.***

The approach described above has the effect of both introducing inter-subject variability and ensuring that the mean of the variability distribution for risk is estimated correctly in each sector.

To better understand the application of Equation 9 in Equations 1 and 2, consider an individual selected at random from a sector. The mean risk for that individual is available from the baseline analysis in

which a single estimate of risk is developed for all individuals in a sector; this mean then is converted to a median using Equation 7. The risk to that randomly selected individual then is

$$\text{Risk}_{\text{individual}} = \text{Risk}_{\text{CT}} \times (\text{IR/BW})_{\text{ratio}} \times \text{ED}_{\text{ratio}} \times \text{CF}_{\text{ratio}} \quad (10)$$

for dioxin exposures and

$$\text{HQ}_{\text{individual}} = \text{HQ}_{\text{CT}} \times (\text{IR/BW})_{\text{ratio}} \quad (11)$$

for mercury exposures where

$\text{Risk}_{\text{individual}}$	=	risk to an individual selected at random from a sector
$\text{HQ}_{\text{individual}}$	=	hazard quotient for an individual selected at random from a sector
Risk_{CT}	=	median risk for individuals in that sector (from the baseline analysis)
HQ_{CT}	=	median hazard quotient for individuals in that sector (from the baseline analysis)
$(\text{IR/BW})_{\text{ratio}}$	=	ratio of the ingestion rate per unit body mass for the individual over the median ingestion rate per unit body mass (selected at random from the normalized distribution for this factor)
ED_{ratio}	=	ratio of the exposure duration for the individual over the median exposure duration (selected at random from the normalized distribution for this factor)
CF_{ratio}	=	ratio of the correction factor for crossing age groups for that individual over the median correction factor (selected at random from the normalized distribution for this factor).

Estimating the variability of $\text{Risk}_{\text{individual}}$ and $\text{HQ}_{\text{individual}}$ in a sector then requires development of variability distributions for $(\text{IR/BW})_{\text{ratio}}$, ED_{ratio} and CF_{ratio} . These are described below.

For each of the parameters displaying variability, separate figures are provided here for the cumulative distribution function (Figures 1 through 5). Comparisons between the data and distributional fits are provided in Tables 1 through 5, and distributional characteristics are summarized in Table 6. For these cumulative distribution functions, the associated probability density functions have not been provided since the original data tables in the *Exposure Factors Handbook* (the source of the data used here) were already in the form of cumulative frequency results. The PDF for a distribution is not actually used in Monte Carlo sampling; instead, it must be converted to a CDF. As a result, only the CDF is relevant for the present analysis and is reported. The locations of medians and means for each distribution of ratios (discussed above) can be found in Table 6, which contains an equation for estimating the mean of any of the ratio distributions from the median ratio and the GSD reported in that table.

Ingestion Rate per Unit Body Mass

This exposure factor component applies to both dioxin and mercury exposures. Variability distributions were developed for ingestion of beef, milk, and fish, and for the age groups 0-5 years, 6-11 years, 12-19 years, and 20+ (adult) years. Since the ingestion rate per unit body mass reported in the *Exposure*

Factors Handbook uses individual-specific body masses when analyzing available data, there is no need to account further for variability in body mass. In other words, the reported variability in the ingestion rate per unit body mass already incorporates inter-individual variability in both ingestion rate and body mass.

Beef Ingestion

The variability of the ingestion rate per unit body mass for home produced beef was determined from the summary of data in Table 13-36 of the 1997 *Exposure Factors Handbook* (1997 EFH), which is entitled “Intake of Home Produced Beef (g/kg-day).” The numerical value associated with each percentile (for a given age group) was first divided by the median (50th percentile) value to obtain $(IR/BW)_{ratio}$. The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 1); on such paper a lognormal distribution appears as a straight line. The geometric standard deviation was determined from the best fitting line by dividing the 84th percentile by the 50th percentile. This procedure was followed for the 6-11, 12-19, and 20+ age groups since data were available in the table for those groups. No such data had been reported for the 0-5 year age group. As a result, the data for the 6-11 year age group were used as surrogate data for the 0-5 year age group in estimating the GSD for the latter group (although the mean for the 0-5 year age group was taken from the data on that age group, and not from the data on the 6-11 year age group as discussed in Section 6.3.1 of the Background Document). The decision to use the 6-11 year age group as the surrogate for the 0-5 year age group in estimating the measure of variance (GSD) was based on the findings in Figure 1, which indicate that the GSD varies as a function of age. The resulting distributional characteristics for all age groups are shown in Table 1.

Milk Ingestion

The variability of the ingestion rate per unit body mass for home produced milk was determined from the summary of data in Table 13-28 of the 1997 *Exposure Factors Handbook* (1997 EFH), which is entitled “Intake of Home Produced Dairy (g/kg-day).” This table summarizes data on intake of all home produced dairy products as averaged over all regions of the country. It is the most complete data set on variability for this exposure pathway, although it requires the assumption that the GSD is the same across all age groups (since only results aggregated across age groups were reported). It also requires the assumption that the variability in fluid milk consumption is the same as the variability in total dairy consumption (data in the 1997 EFH indicates that variability in the former is lower than in the latter for the youngest age group, so variability of milk consumption may have been overestimated in the present analysis for this age group).

The numerical value associated with each percentile (for the aggregate population) was first divided by the median (50th percentile) value to obtain $(IR/BW)_{ratio}$. The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 2); on such paper a lognormal distribution appears as a straight line. The geometric standard deviation was determined from the best fitting line by dividing the 84th percentile by the 50th percentile. The same variability distribution is applied to all age groups (with the GSD values the same for all ages, but the means taken from the age-specific values). The resulting distributional characteristics for all age groups are shown in Table 2.

Fish Ingestion

The variability of the ingestion rate per unit body mass for recreational fish was determined from the summary of data in the final column of Table 12-34 of the 1996 *Exposure Factors Handbook* (1996 EFH), which is entitled “Distribution of Usual Fish Intake Among Survey Main Respondents who Fished

and Consumed Recreationally Caught Fish.” This table summarizes data on intake of fish caught through recreational activities as aggregated over all ages. It is the most complete data set on variability for this exposure pathway, although it requires the assumption that the GSD is the same across all age groups (since only results aggregated across age groups were reported).

The numerical value associated with each percentile (for the aggregate population) was first divided by the median (50th percentile) value to obtain $(IR/BW)_{ratio}$. The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 3); on such paper a lognormal distribution appears as a straight line. The geometric standard deviation was determined from the best fitting line by dividing the 84th percentile by the 50th percentile. The same variability distribution is applied to all age groups, with the means taken from data in the EFH on the different age groups (see Section 6.3.1 of the Background Document). The resulting distributional characteristics for all age groups are shown in Table 3.

Treatment of the Loss Factors

The data described above for beef, milk, and fish reflect the amount of each food category used. They do not reflect the fact that some of the food (particularly in the case of beef) is lost during preparation for eating. The data in the 1997 *Exposure Factors Handbook* on loss during preparation was examined using Table 13-5, entitled “Percent Weight Losses from Preparation of Various Meats.” The data in this table reflect variability across separate instances of food preparation and are based on a random sample from a population similar to that examined in the present analysis (so the issue of surrogate data is not significant here). An individual exposed over many years will prepare the food many times, with intra-subject variability between instances of preparation. This will tend to cause an individual’s lifetime average food loss to converge onto the mean food loss for the population (the issue of convergence to the mean in sampling). As a result, inter-subject variability of the loss factor must reflect the variability of the mean loss factor for the exposed population, not the variability of individual instances of food preparation (the latter variability being much larger than the former for the reasons given above).

To determine the inter-subject variability in the mean loss factor, the distribution of loss factors from Table 13-5 was used as the basis of a Monte Carlo sampling. A sample of size 100 (a reasonable estimate of the number of food preparation events over which the loss factor might be averaged in a person’s exposure duration) was selected at random from this empirical distribution (no distributional shape was assigned *a priori*) and the mean determined for this sample. A second random sample of 100 then was taken and the mean estimated. This process was repeated 2,000 times, yielding a sample of the variability of this mean. The GSD associated with this distribution was less than 1.1, indicating that the loss factor is not a significant source of inter-subject variability relative to the factors described above. It should be noted, however, that the inter-subject variability in loss factor may have been underestimated here since it was assumed that an individual had a loss factor for each instance of food preparation that was drawn randomly from the distribution of loss factors. In actuality, a given individual may consistently lie at the upper or lower ends of the distribution. ***No further consideration of the loss factor was used in the analysis due to the results of this analysis of the relative contribution of different sources of variability, which indicates that this source of variability is insignificant compared to the other sources.***

Exposure Duration

Exposure duration was taken to be equal to the occupancy period in a home. For the farming populations (for which this factor is relevant), data in Table 15-164 of the 1997 *Exposure Factors Handbook*, entitled “Total Residence Time Corresponding to Selected Values of R(t) by Housing Category,” were

used to determine variability in exposure duration for this population. This table contains a statistical summary of such data for all age groups. The variability for the 0-5 year age group was taken from the data on 3-year-olds, since this is the age at the midpoint of the age interval. The variability for the 6-11-year age group was taken from the data on 9-year-olds, since this is the age at the midpoint of the age interval. The variability for the 12-19-year age group was taken from the data on 15-year-olds, since this is the age at the midpoint of the age interval. The variability for the adult age group was taken from the data on 42-year-olds, which is the median age of the adult population.

The numerical value associated with each percentile (for an age group) was first divided by the median (50th percentile) value to obtain ED_{ratio} . The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 4); on such paper a lognormal distribution appears as a straight line. The geometric standard deviation was determined from the best fitting line by dividing the 84th percentile by the 50th percentile. The same variability distribution is applied to all age groups. The resulting distributional characteristics for all age groups are shown in Table 4.

To determine the age-specific median values of ED for the farming population, the data on the non-farming population was used. The decision to use the median values from the non-farming populations as a surrogate for the median values of the variability distribution for the farming population is based on the observation that the age-adjusted median in the farming population is approximately the same as the age-adjusted median in the non-farming population (both are approximately 10 years). The sole difference in these two distributions (farming and non-farming) lies in the variance.

The numerical value associated with each percentile (for an age group) was first divided by the median (50th percentile) value to obtain ED_{ratio} . The distribution of this ratio then was plotted on log-probit paper using the maximum likelihood estimate (see Figure 4); on such paper a lognormal distribution appears as a straight line. The geometric standard deviation was determined from the best fitting line by dividing the 84th percentile by the 50th percentile. The same GSD is applied to all age groups, with the median value for the separate age groups taken from the distributions for the non-farming population as described above. The resulting distributional characteristics for all age groups are shown in Table 4. The procedure for generating these distributions was:

- # The ratio of the median occupancy period for an age group in the non-farming population was divided by the median for the age-adjusted non-farming distribution.
- # The same ratio was assumed to hold for that age group in the farming population. The median of the age-adjusted distribution for the farming population was multiplied by the ratio obtained in the first step for the same age group.
- # For this same age group in the farming population, the GSD of the distribution was assumed to be the GSD of the age-adjusted distribution, based on the fact that the GSD was not a significant function of age in the non-farming population.

Correction Factors for Crossing Age Groups

The central tendency calculations employed the assumption that an individual beginning exposure at age X continued exposure under parameter values identical to those of the age group in which the age X falls. This means the ingestion rate per unit body mass was constant during the exposure interval even if, in reality, that individual would have crossed into a higher age group at some point during the exposure interval. Since the ingestion rate per unit body mass generally decreases with age, particularly in the first

several age groups, the central tendency estimates will tend to overestimate slightly the mean values of risk.

To explore the effect of this assumption used in the central tendency estimates, the calculations of lifetime risk were repeated using a life-table approach in which individuals were followed year-by-year throughout the period of exposure. (*Note: the term life-table also is used in the epidemiological literature to refer to probabilities of survival at specific ages. This is not the usage here*). During each year, age-specific intake rates per unit body mass were used, with these changing as the individual ages. A Monte Carlo procedure was developed in which:

- # An age at beginning of exposure was selected at random (uniform distribution) from within an age interval (e.g., the 0-5 year age interval).
- # An exposure duration was selected at random based on the distribution described previously (specific to that age interval in which exposure began).
- # The life-table information was used to calculate the total ingestion per unit body mass (g/kg) over the selected exposure period assuming the ingestion rate per unit body mass changes with age.
- # The total ingestion per unit body mass (g/kg) was calculated for the same exposure duration assuming the ingestion rate per unit body mass did not change (i.e., the assumption of the central tendency calculation).
- # The ratio of the total ingestion per unit body mass using the life-table over the total ingestion per unit body mass calculated without the life-table was obtained.
- # This process was repeated over a sample of size 1000 for the same age interval (e.g. 0-5 year age interval).

The result of this process is a sample of 1,000 calculations of the ratio of the lifetime intake per unit body mass with a life-table approach and without such an approach. This, in turn, is equivalent to a sample of 1,000 ratios of the “true” lifetime intake per unit body mass over the value obtained by the central tendency approach. The median for this population of samples is 1.0 (so the original assumption used in the central tendency estimates did not produce a biased result). The GSD was obtained by plotting this distribution on log-probit paper (see Figure 5), determining the maximum likelihood fit, and determining the ratio of the 84th percentile over the 50th percentile. Separate distributions were developed for each of the four age groups. Results are summarized in Table 5. This correction factor was applied only for estimation of cancer risks (i.e., TCDD calculations for beef and milk ingestion) and not for fish ingestion (i.e., methylmercury calculations), since it is irrelevant in the case of non-cancer effects.

VI. Analytic Methodology

The methodology of Monte Carlo analysis employed here to assess variability is structured to follow the guidelines set out in EPA's *Guiding Principles for Monte Carlo Analysis* (EPA, 1997). To incorporate variability of exposure into the distributions Prob_{var} and HQ_{var} from Equations 1 and 2, the present assessment returns to the stage in calculations at which the sector-constituent-pathway-specific exposures are calculated; i.e., the stage at which $S_{imn} \times DC_{ijmn} \times EF_{ijmn}$ is calculated in Equations 1 and 2. This point estimate from the baseline assessment then is converted to a median value as explained in Section V. Normalized variability of EF_{ijmn} within the sector then is characterized for each constituent and exposure

pathway using the methods described previously. Monte Carlo analysis then is performed using Equations 1 or 2 (depending on the constituent), with the probability of sampling an individual from a given sector being equal to the fraction of the exposed population in that sector. Once an individual is selected from a sector, random values for EF_{ijmn} are selected by multiplying the median EF_{ijmn} by randomly selected values from the normalized distribution EF_{ratio} as described in Section V. This process is repeated over all sectors, resulting in a composite variability distribution for either $Prob_{var}$ or HQ_{var} which is specific to an age group and receptor population.

Note that for the receptor populations and constituents considered in this analysis, there is a single pathway and constituent which dominates (see the discussion in Section II). The calculation of variability distributions is simplified when there is a single constituent and a single exposure pathway in Equations 1 and 2. If this is the case, a single distribution of EF_{ij} must be generated and used in the process above, rather than one for each of several combinations of pathways and constituents contributing to the risk in individuals. There then is no summation over constituents and exposure pathways, resulting in simplified versions of Equations 1 and 2:

$$Risk_{ij} = S_i \times DC_{ij} \times EF_{ij} \times SF_{ij} \quad (12)$$

$$HQ_{ij} = S_i \times DC_{ij} \times EF_{ij} \times SF_{ij} \quad (13)$$

The present assessment of variability uses this simplified approach for a subset of receptor populations where there is a dominant compound and exposure pathway (identified in Section II). Note that HI in Equation 2 is replaced by HQ above since there is a single constituent considered.

The HWC risk analysis first generated one mean risk estimate for each:

- # Facility (the number of facilities depends on the facility category)
- # Sector surrounding a facility (16 per facility)
- # Compound or constituent (dioxin or mercury)
- # Age group (0-5, 6-11, 12-19, adult)
- # Relevant receptor subpopulation (beef cattle farmer, dairy cattle farmer, or recreational fisher).

For example, if there were 10 facilities, there would be 160 sectors and for each of these 160 sectors there would be a single central tendency risk estimate for dioxin exposures to dairy cattle farmers in the 11 to 19-year-old age group. This example will be used in the following discussion; exactly the same methodology was applied to all facility category/constituent/receptor analyses.

In the present study, variability in exposure characteristics (EF in Equations 12 and 13) is incorporated into the analysis after these separate sector-specific central tendency risk estimates have been generated. As a result, the present assessment is referred to as “post-processing” since the variability is incorporated after the point, mean, estimates of risk have been generated in each sector. This post-processing approach is valid because the values S_i , DC_{ij} and S_{ij} in Equations 12 and 13 are not treated as variable within a sector, and all individuals in a sector have values of EF selected from the same variability distribution.

In the example above, there are 160 mean estimates of the risk generated in the baseline analysis (one for each sector). For each of these 160 central tendency estimates, a lognormal variability distribution for

EF_{ratio} with a median of 1.0 and an associated geometric standard deviation was assigned based on some combination of the factors mentioned in Section V (variability of ingestion rate per unit body mass, exposure duration, and correction factor for crossing age groups in the case of dioxin exposures; variability of ingestion rate per unit body mass in the case of mercury exposures). This distribution of EF_{ratio} then was multiplied by the median value in a sector (obtained by converting the mean to a median as described in Section V) to yield the distribution of risk in that sector. This resulted in 160 separate variability distributions for the example used here, each describing variability of risk within a sector.

The task is to combine these 160 separate variability distributions into a single, composite, variability distribution. This composite distribution must weight the 160 separate distributions according to their relative contribution to the total population; i.e., the contribution of a given sector's variability distribution to the composite variability distribution must equal the fraction of the total exposed population contained in that sector (where this fraction depends on the receptor subpopulation). While the separate distributions are lognormal, the weighted sum of lognormal distributions is not itself lognormal. As a result, there is no analytic solution to the statistical characteristics of this composite distribution. A Monte Carlo sampling procedure based in the software CrystalBall® was used, therefore, to construct the composite distribution (this software is one of the risk assessment profession standards for performance of variability and uncertainty analyses). The steps of sampling are as follows:

- # The population size in each separate sector was determined from GIS analysis.
- # The total size of the exposed population was determined by summing populations across all sectors in the assessment.
- # The fraction of the total exposed population contained in each sector was calculated by dividing the population in a sector by the total exposed population.
- # A random number was generated using a uniform probability density on the interval [0,1]; the algorithm for this generation was the RAND() function in EXCEL (CrystalBall® resides on top of EXCEL).
- # A sector was selected at random from the total population of 160 sectors using this random number. The probability of a particular sector being selected was equal to the fraction of the total exposed population in that sector (see the discussion in Crawford-Brown, 1996).
- # Once a sector was selected at random, the variability of EF_{ratio} in that sector was assigned as a lognormal probability density function with median of 1.0 and geometric standard deviation specific to that constituent, pathway and receptor population (see Table 1).
- # One sample of the value of EF_{ratio} was selected from the variability distribution using Monte Carlo sampling with a seed value of 0.0. This value of EF_{ratio} was multiplied by the median value for risk in that sector to obtain the value of risk for that sampled individual; this was stored in a file (a "forecast" file within CrystalBall®).
- # The process above was repeated for the number of trials necessary to meet criteria of stability for the resulting composite variability distribution (see the discussion later in this section).
- # Risks associated with prescribed percentiles of the composite variability distribution (e.g. 50th, 75th and 97th percentiles) then were determined.

Truncation of Distributions

It is common in fitting variability data to find that the distributions are partially truncated at the lower and upper ends of the distribution, with truncation usually at between 2 and 3 geometric standard deviations (Crawford-Brown, *Theoretical and Mathematical Foundations of Human Health Risk Analysis*, Kluwer Academic Publishers, 1997). This is due to physical and biological limitations on the range of values that can occur. This general result was found to hold in the present analysis, as the data from the *Exposure Factors Handbook* could be fitted appropriately by a lognormal distribution out to approximately 2 to 3 geometric standard deviations around the median. Beyond that range, the lognormal distribution is inaccurate (as are all analytic, *a priori*, distributional forms) since the probability density for the data outside this region is significantly less than that predicted by the distribution. In the present analysis, truncation for sampling was at 3 geometric standard deviations; values beyond these limits were rejected and resampled. This truncation is not shown in Figures 1 through 5. *To reflect truncation, the reader can follow the displayed curves to approximately the 1% and 99% values at the two tails, and then draw lines horizontal to the X-axis from these two points.*

Note: Truncation does not introduce inaccuracies into the composite risk or HQ variability distribution for the population. As mentioned above, truncation is a feature of the underlying data on which the parameter variability distributions are developed, and are not introduced a priori into the analysis. Failure to truncate the lognormal distributions would introduce inaccuracies by artificially increasing the likelihood of parameters being selected at values more than 3 GSDs from the median.

Sampling Criteria for the Cumulative Probability Distributions

A trade-off is necessary in selecting the sample size for a Monte Carlo analysis. A larger sample size improves the estimates of risk associated with each percentile in the variability distribution. This larger sample size, however, requires greater computation time, with the potential for computation times that are too long to provide timely answers for decisions. The number of samples employed in the Monte Carlo analysis performed here was based on criteria related to the stability of the median (50th percentile) risk value and of the 97th percentile, following guidelines in EPA's *Guiding Principles for Monte Carlo Analysis* (EPA, 1997). Sample size was selected initially to be 1,000 runs of the model (i.e., 1,000 randomly selected individuals from the exposed population). Sample size then was increased in increments of 500 on the same model, and estimates at the 50th and 97th percentiles compared (e.g., the median estimate for a sample size of 1,500 compared against that for a sample size of 1000, and the 97th percentile estimate also compared at these two sample sizes). Sample size was increased until the change in the estimate (for both the 50th and 97th percentiles) was not larger than 5%. ***This criterion ensures the stability of the first decimal place of the percentile estimates, which is consistent with the number of significant digits available through the underlying data sets.***

For example, if the 97th percentile value of the risk were 1×10^{-6} with a sample size of 3000, this sample size was considered adequate if and only if the 97th percentile value of the risk for a sample size of 2,500 was between 0.995×10^{-6} and 1.05×10^{-6} . If it were not, the sample size would be increased to 3,500 and the run performed again. This test was run on several of the facility categories with the largest number of facilities (where meeting the criterion would be most difficult). In particular, it was run for the facility categories with more than 200 facilities. From this analysis, it was determined that a sample size of 3,000 runs provided the necessary stability of the variability distribution at both of these percentiles. This sample size then was used in all analyses.

Treatment of the Recreational Fishing Scenario

As described in Section II, the question asked for recreational fishers was: *for which facilities may it be stated that some fraction of the recreational fishers exposed in the sectors surrounding that facility have a hazard quotient (HQ) greater than or equal to 1.0, and if there are an equal number of recreational fishers in all sectors, what is the variability of HQ in the total exposed population of recreational fishers?* To answer this first question, the variability distribution of hazard quotients from methylmercury exposures via fish consumption within each sector was generated. The 95th percentile of this variability distribution then was examined in each sector. Any sector where this 95th percentile estimate is above 1.0 was “flagged” as being of potential concern. Note that this analysis does not identify the **number** of individuals exceeding this hazard index, and does not produce a population-weighted variability distribution for a facility category.

To address the second question, the analysis of variability used for the dioxin exposures was repeated for the methylmercury exposures, with the exception that the population in each sector was equal. As a result, the probability of selecting an individual from a given sector was the same for all sectors. The result is the variability distribution of hazard quotients in a hypothetical population spread evenly across the geographic sectors.

Relationship Between Central Tendency Estimates of Risk and Median Estimates of Risk from the Variability Analysis

Two analyses were performed of the risks to individuals in the exposed populations. In the first analysis (conducted prior to the variability analysis discussed in this report), point, mean, estimates of exposure parameters were used in Equations 3 and 4, and the distribution of risk in the total exposed population generated without incorporating variability of exposure parameters.

In the second analysis, described in this report, variability distributions were developed to reflect variability of risk as introduced by variability in exposure factor parameters. Since the median value for the distribution of EF_{ratio} is identical to the adjusted median value used in the first analysis (i.e. where the mean from the first analysis is converted to a median as described in Section V), it might be assumed that the median value of the distribution showing variability of risk when parameter variability is incorporated would be the same as the adjusted median value of risk when parameter variability is not incorporated. In addition, it might be assumed that the mean in both analyses should be the same.

This is not, however, the case, as can be seen from the results of this analysis. The median of the variability distribution for risks associated with a facility category when exposure factor variability is incorporated differs from the median of the variability distribution produced without consideration of exposure factor variability. The same is true for the means. The reason for this apparent discrepancy lies in the fact that multiple facilities, and multiple geographic sectors or sub-regions surrounding a facility, are combined within the analysis of risks for a facility category. When lognormal variability distributions are constructed around point estimates in individual sectors, and these separate sector-specific distributions combined (as in the present analysis), the composite variability distribution will not be lognormal and the median of that composite variability distribution will not be the same as the median of the distribution produced when exposure factor variability is ignored. The same is true for means. This would not have been the case if the exposure factor variability distributions had been symmetrical, but they are not.

As an example of the reason for this difference, let R_{ij} be the median estimate of risk for a given age group and subpopulation (e.g., adult recreational fisher) in sector i associated with facility j (there being

N such facilities in a facility category). Consider two such sector and facility combinations (e.g., $R_{3,8}$ and $R_{7,5}$). Let the median value of $R_{3,8}$ be 1×10^{-5} and of $R_{7,5}$ be 1×10^{-4} . Let 30% of the population be found in sector 3,8 and 70% in sector 7,5. Let the geometric standard deviation for the variability distributions associated with the relationship between exposure and risk be 4.0 for both sectors (the medians of the variability distributions are the central tendency estimates).

Without exposure factor variability, the median for this situation is 1×10^{-4} (since 30% of the population is at a risk of 1×10^{-5} and the remaining 20% needed to reach a cumulative fraction of 50% is at 1×10^{-4}). Note that there are no individuals with a risk of between 1×10^{-5} and 1×10^{-4} . With exposure factor variability incorporated, the medians for the two separate populations do not shift (these are still 1×10^{-5} and 1×10^{-4} for the first and second populations, respectively). Some of the individuals from the first population, however, will have a risk above 1×10^{-5} , and some from the second population will have a risk of below 1×10^{-4} . The result is that risk values between 1×10^{-5} and 1×10^{-4} will now be present in the composite distribution of risk. In this example (simulated for the present analysis using Monte Carlo analysis), the median of the composite distribution shifts to 5.1×10^{-5} . Both the degree and the direction of shift of the median (from the value that is present without variability incorporated) are functions of the difference in central tendency values for the two distributions, the geometric standard deviations for the variability distributions, and the fraction of the total exposed population contained in each of the two distributions.

VII. Uncertainty Analysis Using SUDAAN

The structure of the analysis for risks associated with specific facility categories divided the risk assessment into several stages:

- # First, the central tendency risk was calculated for the population in each separate geographic sector surrounding each facility sampled from the facility category. This produced a mean risk for individuals in each sector.
- # Second, the variability of risk associated with inter-subject variability of exposure parameters was calculated for the population in each separate sector surrounding each facility sampled from the facility category. This produced a point estimate of the risk associated with each specific percentile from that sector's variability distribution.
- # Third, the variability of risk in the total exposed population was determined by aggregating the variability distributions from different sectors, weighted by the population in that sector, into a composite distribution. This produced a point estimate of the risk associated with each specific percentile from that aggregate distribution describing the total exposed population.
- # Finally, the uncertainty associated with specific percentiles in this aggregate distribution was determined and summarized as confidence intervals around the point estimates of the risks associated with each percentile produced in the third step.

The first through third steps of the analysis have been described in previous sections. This section focuses on the fourth step, and on the validation of this step through use of the Monte Carlo variability analysis as a benchmark. The goal of this step was to construct confidence intervals around the point estimates of specific percentiles in the variability distribution for the aggregate, exposed population. The procedure for estimating these confidence intervals was the SUDAAN software. SUDAAN allows specification of a single value of the risk associated with each geographic sector, and so is able to reflect both inter-facility and inter-sector variability of the environmental concentrations. This, in turn, allows

the calculation of variances associated with the selection of a subset of facilities from a facility category. SUDAAN does not, however, explicitly handle variability distributions within a sector such as those that arise from inter-subject variability of exposure parameters. Without this latter source of variability incorporated into the SUDAAN analysis, the estimates of variance produced by SUDAAN do not include variance introduced by sampling of individuals from the variability distribution in a sector.

It is possible, however, to reflect inter-subject variability within a sector through the use of a discrete approximation to the inter-subject variability distribution within that sector, with each interval of the discretized variability distributions treated as a subsector within the sector. This discrete approximation introduces a potential error into the calculation of both the best estimate values and confidence intervals for the risk associated with specific percentiles in the aggregate variability distribution. It is necessary, therefore, to determine whether the approach used to generate confidence intervals around point estimates of percentile values in the variability distribution accurately captures the best estimates of these percentiles. This following discussion describes both how the aggregate distribution was determined and how the accuracy of the approach based on discrete distributions and SUDAAN was assessed.

VII. Discrete Distributions and SUDAAN

As described above, SUDAAN produces an estimate of the confidence intervals around specific percentiles in the variability distribution for the total exposed population (total aggregated over all sectors). To do this, it is necessary to provide as input into SUDAAN a single value of risk for each sector around a facility. If a single value is provided for each geographic sector, the analysis of variance reflects uncertainties introduced by:

- # Sampling a subset of facilities from within the set of all facilities
- # Inter-facility variability in source characteristics and environmental parameters
- # Inter-sector variability of environmental concentrations due to dispersion patterns around a given facility.

These three contributions to the variance in risk estimates for the exposed population, however, omit the variance introduced by inter-subject variability of risk within each sector. The result can be errors in both the point estimates of percentiles in the aggregate variability distribution and in the confidence intervals constructed around these point estimates.

To reflect the influence on the variance contributed by inter-subject variability within a sector, the SUDAAN analysis divided the inter-subject variability distribution within each geographic sector into a series of subsectors (such subsectors can be accommodated within SUDAAN), each characterized by a single value of risk for the individuals contained in that subsector. Each subsector represents a subset of the population contained within a sector, rather than representing a geographic subdivision of the sector (i.e., the individuals in different subsectors of a sector are at the same geographic location; they differ only in representing different exposure factor values). The result is a discrete approximation (histogram) of the continuous variability distribution of risk for that sector. In the analysis performed here, the variability distribution in a sector was divided into 20 subsectors, each containing 5% of the total population within that sector (SUDAAN must have the population size equal within the subsectors of a given sector).

The task then was to determine a single value of the risk to be assigned to all individuals in each subsector. This single value is the mean risk for the individuals in that subsector. The requirement that it

be the mean, rather than some other quantity such as the median, arises from the need to retain the correct mean value for the entire population in the sector containing the 20 subsectors. Determination of this mean for a given subsector within a sector requires specification of the lower and upper endpoints within the variability distribution (for the sector) associated with that subsector, conditional upon the requirement that each subsector contain 5% of the total population in a sector.

Let $PDF(x)$ be the probability density function (lognormal) for the risk in a sector; $PDF(x)$ reflects the inter-subject variability of risk in that sector. $PDF(x)$ must then be divided into 20 subsectors of equal cumulative probability (i.e. 0.05). For the first subsector, the condition is that the integral of $PDF(x)$ from 0 to UL (the upper limit of the first subsector) must equal 0.05. The lower limit (LL) of the second subsector is the value of UL for the first subsector. This process is continued through all 20 subsectors.

Determination of the lower (LL) and upper (UL) limits of each of the 20 subsectors was accomplished through numerical integration (Newton's method) of the probability density function $PDF(x)$, since there is no analytic solution to this integral for lognormal PDFs. Let Δx be the size of the integration interval used in the numerical integration of $PDF(x)$. In defining LL and UL for the first subsector, the value of $PDF(\Delta x/2)$ was calculated (this being at the midpoint of the first integration interval). The integral of $PDF(x)$ from 0 to Δx then equals $PDF(\Delta x/2) \Delta x$. If this value is less than 0.05 (which it was in this analysis, since Δx must be small relative to 0.05), the integration continues and $PDF(3\Delta x/2) \Delta x$ is added to $PDF(\Delta x/2) \Delta x$. Again, this is compared against 0.05 and, if it less than 0.05, the integration continues and $PDF(5\Delta x/2) \Delta x$ is added to $PDF(\Delta x/2) \Delta x$ plus $PDF(3\Delta x/2) \Delta x$. This process continues until 0.05 is reached by the sum. The value of UL for the first subsector in that sector then equals Δx times the number of integration intervals necessary to reach 0.05. This also becomes LL for the second subsector, and UL for that subsector is determined as above.

Once the lower and upper limits were determined for each subsector in a sector, the mean value for the risk in each subsector was determined, again through numerical integration. Let $PDF(x)$ be the probability density function for the risk in a sector, lognormal in shape. The mean risk in a subsector then is the integral of the product $xPDF(x)$, with the integration performed numerically (Newton's method) from LL to UL as described previously (LL and UL are specific to that subsector, and $PDF(x)$ is specific to the sector containing the subsector). This mean value was assigned to all individuals in that subsector, and this mean value was provided to SUDAAN for the analysis of variance (20 such mean values for each sector).

VIII. Comparison of SUDAAN to Monte Carlo

There is a potential error introduced by this approach, since all individuals in a subsector are assigned the mean for that subsector (even though in reality there is a continuous distribution within that subsector). This essentially replaces the continuous distribution by a discrete distribution. The question to be addressed is: ***Does the use of a discrete distribution, with the characteristics described above, cause significant inaccuracies in the best estimates of the percentile values in the variability distribution developed for the total exposed population (i.e., aggregated over all geographical sectors)?*** To address this question, several procedures were developed and applied to a sample of populations as described below.

In the first analysis, the best estimates of the 95th, 97th and 99th percentiles for the aggregated distribution as produced by SUDAAN were compared against the estimates from the Monte Carlo analysis described previously for a sample of the facility categories and control options. The Monte Carlo analysis employs the full distribution in each geographical sector, rather than a discrete approximation of the variability

distribution (but does not characterize uncertainty properly and so is not useful for this uncertainty analysis). The results for all facility categories, and with all control strategies (including baseline) were used for this analysis where both the Monte Carlo and the SUDAAN estimates were available. The ratio of the SUDAAN best estimate result over the Monte Carlo result (for the same percentile of the variability distribution) was obtained, and a distribution developed of these ratios. The mean and standard deviation of this distribution of ratios then was determined. The desired feature here would be a mean of 1.0, indicating that, on average, the Monte Carlo and SUDAAN estimates agreed, and a standard deviation of 0, indicating they agreed at all times.

For the beef farmer, this analysis produced a mean of 0.98 and a standard deviation of 0.15 for the 95th percentile values. For the beef farmer, this analysis produced a mean of 0.91 and a standard deviation of 0.2 for the 97th percentile values. For the beef farmer, this analysis produced a mean of 0.8 and a standard deviation of 0.2 for the 99th percentile values. For the dairy farmer, this analysis produced a mean of 0.97 and a standard deviation of 0.17 for the 95th percentile values. For the dairy farmer, this analysis produced a mean of 0.95 and a standard deviation of 0.15 for the 97th percentile values. For the dairy farmer, this analysis produced a mean of 0.91 and a standard deviation of 0.15 for the 99th percentile values. For the recreational fisher, this analysis produced a mean of 1.02 and a standard deviation of 0.3 for the 95th percentile values. For the recreational fisher, this analysis produced a mean of 1.04 and a standard deviation of 0.3 for the 97th percentile values. For the recreational fisher, this analysis produced a mean of 0.8 and a standard deviation of 0.22 for the 99th percentile values. These six analyses also are summarized in Figures 6 through 13. The reason for plotting these as bar graphs is that it was possible to locate samples (e.g., recreational fishers in the LWAC category with a particular control strategy) where there were systematic differences between the SUDAAN and Monte Carlo results. This allowed a quality assurance check and identified several areas of miscalculations that were resolved before this final analysis was generated. In these figures, RF stands for recreational fisher, BF stands for beef farmer, DF stands for dairy farmer, “new” refers to the SUDAAN results and “old” refers to the Monte Carlo results.

From this analysis, it may be concluded that use of the SUDAAN procedure with the introduction of the discrete approximation for the intra-sector variability distributions (with 20 discrete slices) produces best estimates of the upper percentiles (95th, 97th and 99th) of the aggregate distributions of risk within 25% of the values obtained using the Monte Carlo analysis as the benchmark procedure. For example, if the Monte Carlo procedure (which is the most accurate for estimating variability) indicated that the risk associated with an individual at the 95th percentile of the variability distribution is 10^{-4} , the SUDAAN analysis generally produced an estimate at this same percentile that was between 0.75×10^{-4} and 1.25×10^{-4} . *It should be borne in mind that the results presented above indicate the upper limit on the degree of difference between the Monte Carlo and SUDAAN results, since rounding differences (only the first significant digit was used in the comparison) in the two procedures will produce ratios that are further from 1.0 than would be the case if more significant digits were examined. It is likely that the actual differences are smaller.*

IX. Influence of the Number of Subsectors

In a second analysis, a Monte Carlo procedure for sampling from variability distributions associated with populations (one population from each of two sectors) was developed. Two populations were assumed: one with a mean of 10 and a geometric standard deviation of 4 (lognormal PDF) and a second with a mean of 1 and a geometric standard deviation of 4 (lognormal PDF). The discrete approximation to the variability distributions should introduce the largest inaccuracies into the estimates of the 95th, 97th and 99th percentiles of the aggregate distribution when 100% of the exposed population is in only one of the two distributions described above. As the two distributions become more equal in size, the errors in the 95th, 97th and 99th percentiles should be reduced.

The procedure for this analysis was to first select a fraction of the aggregate population in each of the two distributions. The parameter f is taken to be the fraction in the first population (the population with mean of 10). The fraction in the second population then is $1-f$. A Monte Carlo procedure then was developed to sample at random from each of the two distributions, with the probability of sampling from the first distribution being f and the probability of sampling from the second being $1-f$. A total of 10,000 samples were obtained to construct the aggregate variability distribution of risk for the population (this being the sample size necessary to ensure that the 97th percentile can be estimated to within 5%, the criterion selected for stability of that estimate). The 95th, 97th and 99th percentiles for this distribution were obtained and recorded. This process then was repeated for values of f between 0 and 1.0, in increments of 0.1.

To simulate the SUDAAN analysis, the two distributions then were developed in discrete form through the method of numerical integration described previously. The mean in each “slice” of this discrete distribution was assigned to each individual from that slice, as was done in the SUDAAN analysis. This resulted in N numerical values for each of the two distributions, where N is the number of slices in the discretized distribution. The fraction of people assigned to each slice equals the fraction of people in that population (there are two populations) divided by N ($1/N$ being the fraction of a given population contained in a slice). Monte Carlo analysis then was used (with a sample size of 10,000) to determine the variability distribution for the aggregate population and to specify the 95th, 97th and 99th percentiles of the aggregated distribution.

The 95th percentile from the SUDAAN result minus the 95th percentile from the non-discretized (Monte Carlo) result then was calculated and divided by the 95th percentile from the non-discretized result for each value of f . The same was done for the 97th and 99th percentiles. The magnitude of this ratio (with the absolute value of the differences) indicates the fractional degree of inaccuracy introduced by the discretization of the variability distributions for the two populations. This inaccuracy will become smaller as the number of “slices” is increased (going to 0.0 as N approaches infinity, since the distribution no longer is discretized and the method approaches the Monte Carlo method). Values of N equal to 5, 10, 20, 30 and 40 were examined. The results are summarized in Table 7 (only the limiting cases of f equal to 1 and 0.5 are considered).

In summary, the use of 20 slices for each variability distribution produces estimates of risk associated with individuals at the upper percentiles (95th and 97th) of the aggregate distribution that are within 25% of the values obtained from a complete Monte Carlo analysis. Bear in mind that the actual error introduced decreases as the number of populations sampled to create the aggregated population increases, and that in the actual SUDAAN analysis developed for this project the number of sampled populations was significantly larger than 2. This analysis, therefore, supports the contention that the SUDAAN approach adequately represents the inter-subject variability distribution for risk in the total exposed population, and may be used for both the variability and uncertainty analyses.

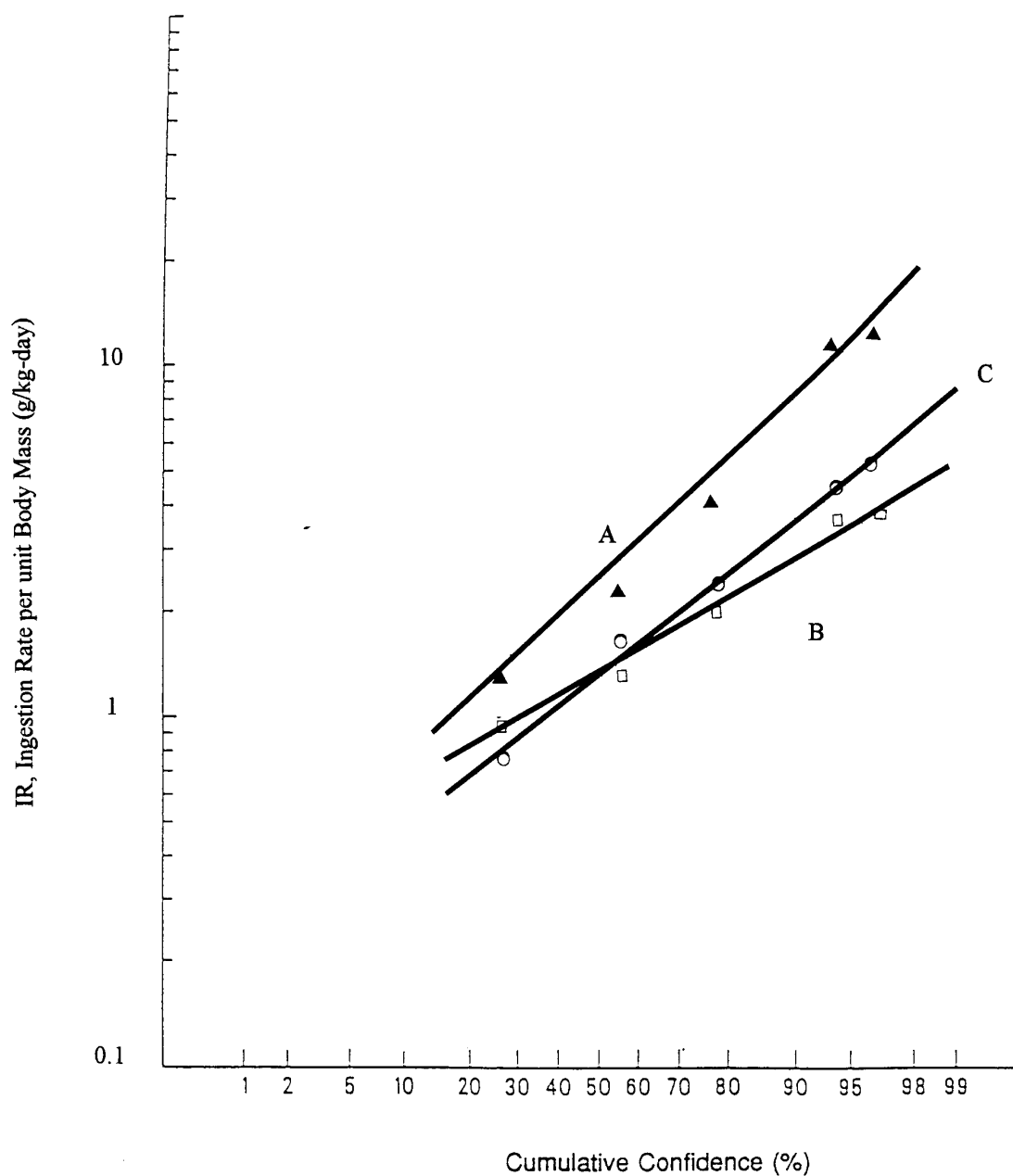


Figure 1. Variability of the ingestion rate per unit body mass for home produced beef. Curve A is for the 6-11 year age group (and the 0-5 year age group; curve B is for the 12-19 year age group. Curve C is for adults. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The geometric standard deviations are the only properties of these curves relevant to the analysis in this memo; they equal the ratio of the 84th percentile over the 50th percentile.

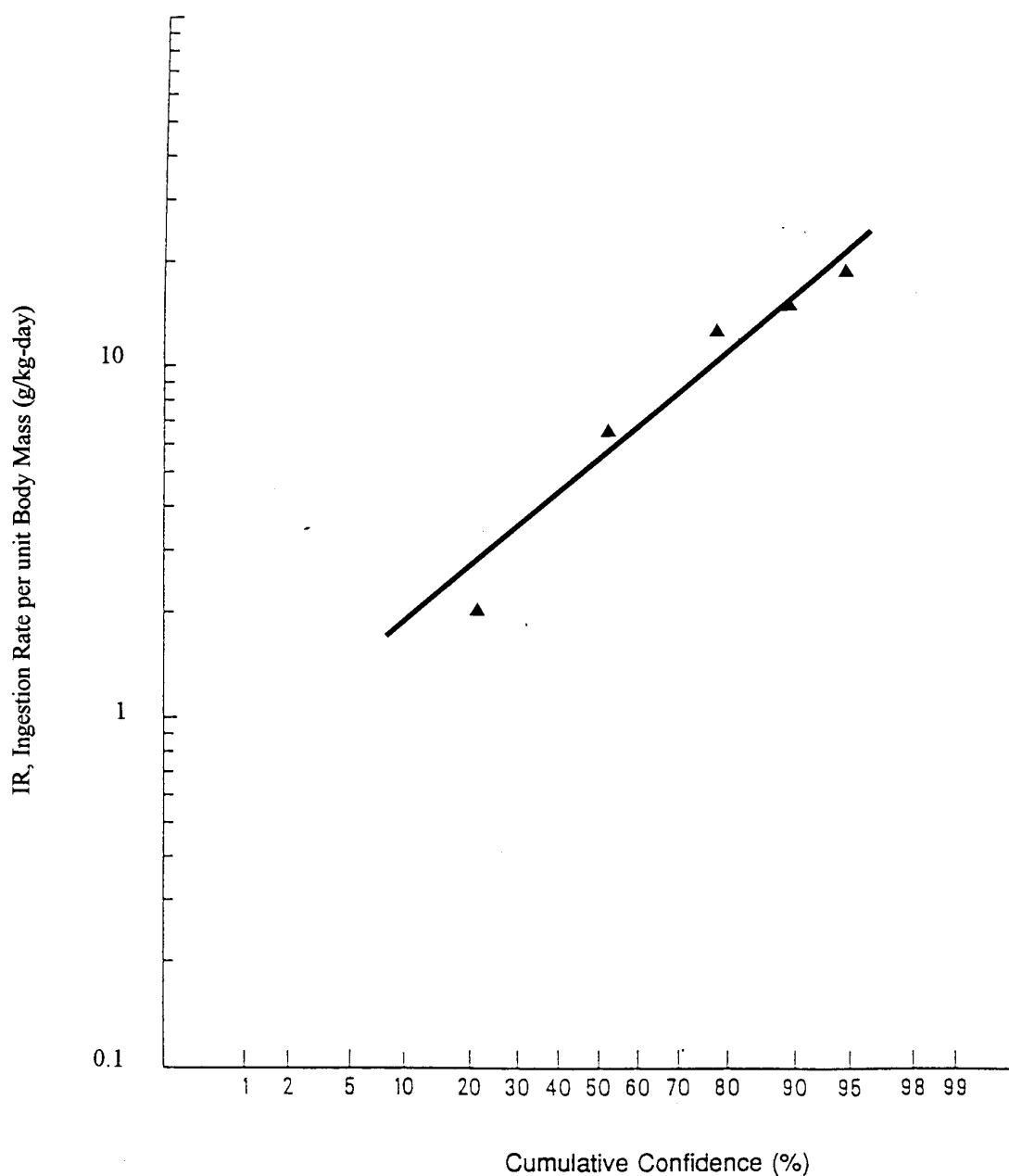


Figure 2. Variability of the ingestion rate per unit body mass for home produced milk. The curve is for all age groups. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The geometric standard deviations are the only properties of these curves relevant to the analysis in this memo; they equal the ratio of the 84th percentile over the 50th percentile.

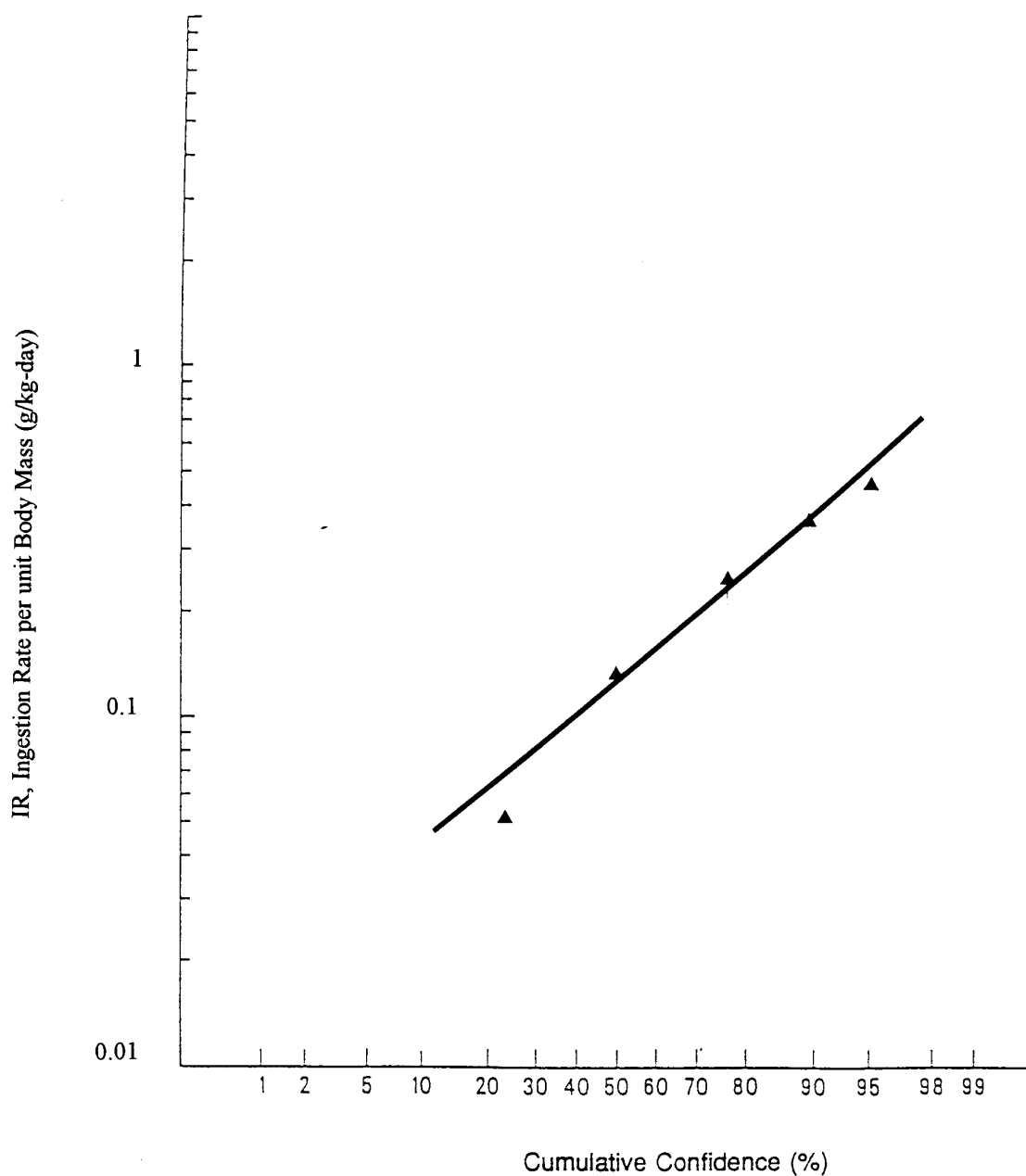


Figure 3. Variability of the ingestion rate per unit body mass for the recreational fisher. The curve is for all age groups. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The geometric standard deviations are the only properties of these curves relevant to the analysis in this memo; they equal the ratio of the 84th percentile over the 50th percentile.

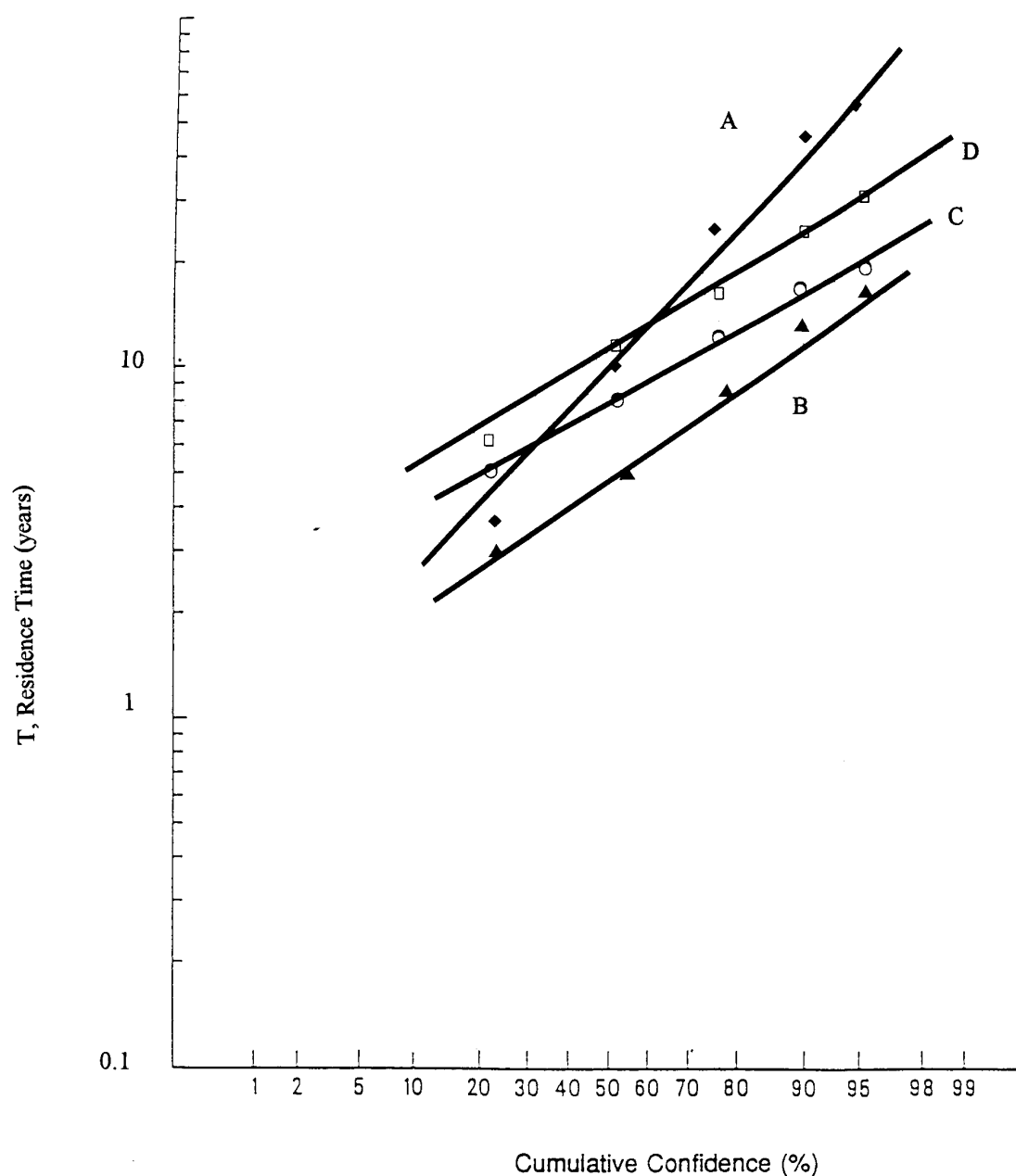


Figure 4. Variability of the residence times (occupancy periods) for the farming and non-farming populations. Curve A is for the farming population (all ages); curve B is for the non-farming population 0-5 years). Curve C is for the non-farming population (6-11 and 12-19 years). Curve D is for the non-farming population (adults). The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis. The geometric standard deviations are the only properties of these curves relevant to the analysis in this memo; they equal the ratio of the 84th percentile over the 50th percentile.

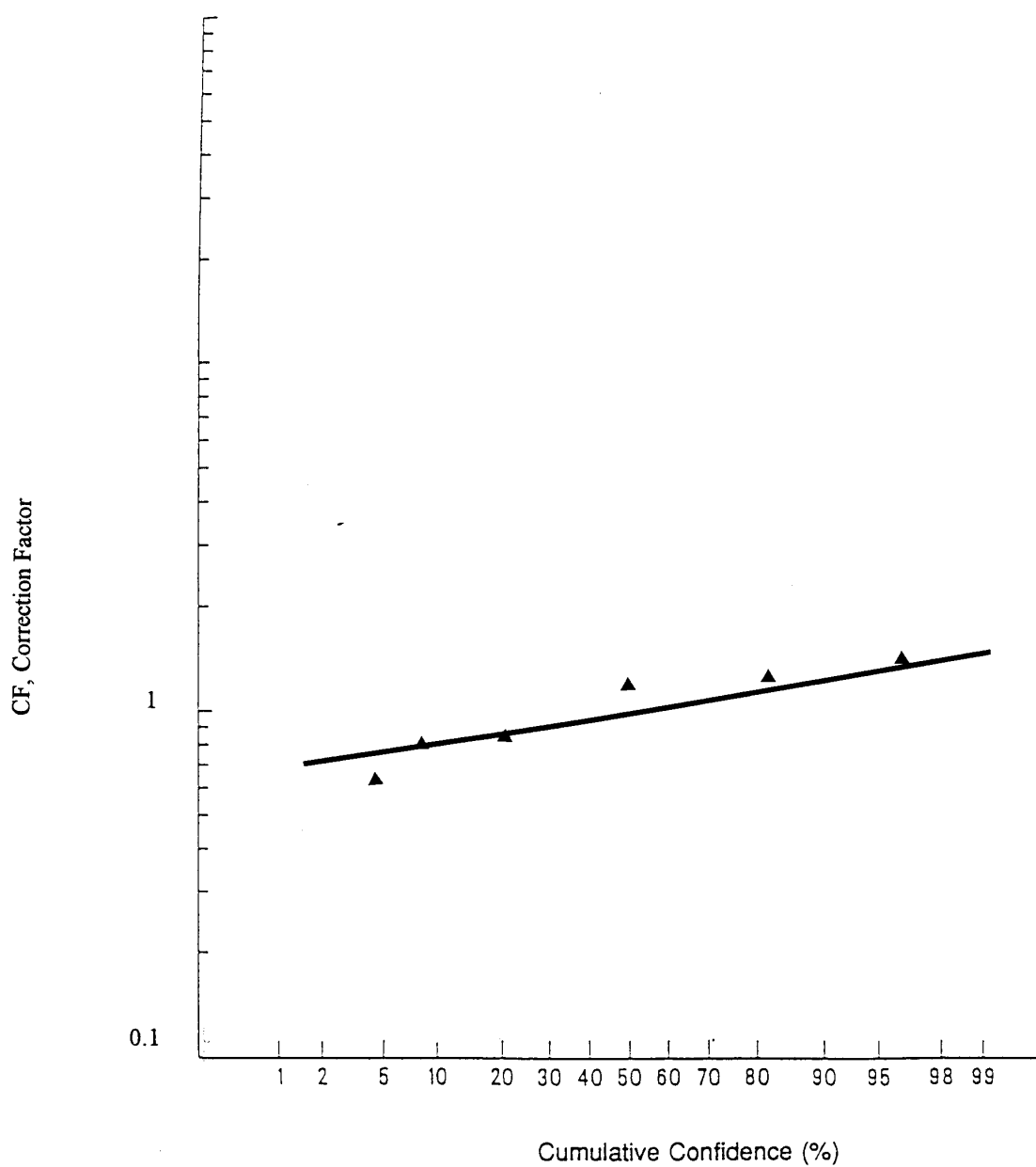


Figure 5. Variability of the correction factor for crossing age groups. There is no significant difference for the 3 exposure pathways, so the same distribution is used. The curve applies to the 0-5, 6-11 and 12-19 year age groups only. The cumulative confidence refers to the fraction of results with a value less than or equal to that shown on the y-axis.

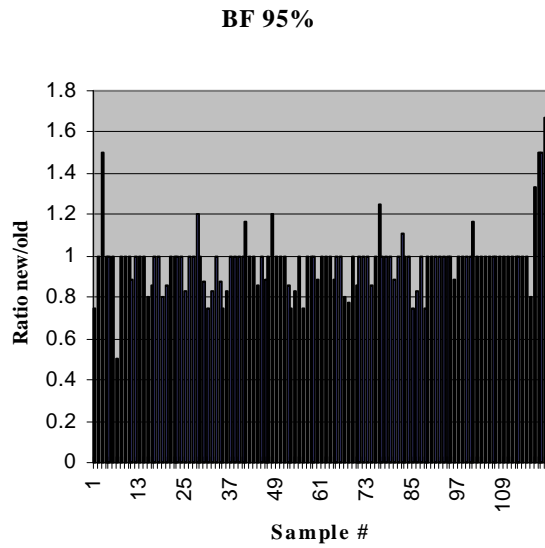


Figure 6.

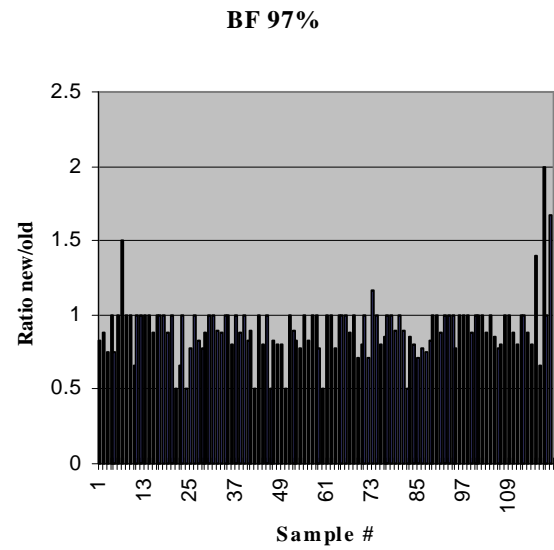


Figure 7.

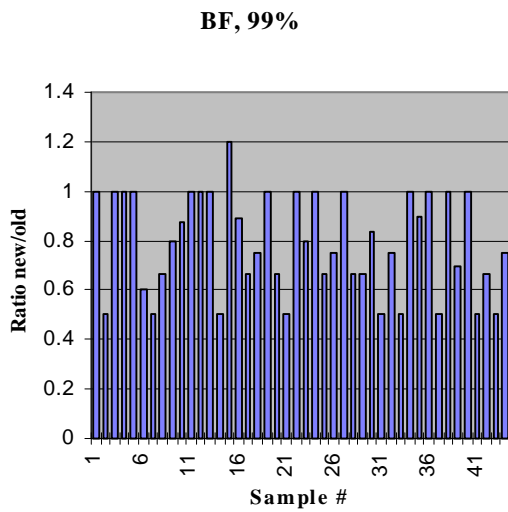


Figure 8.

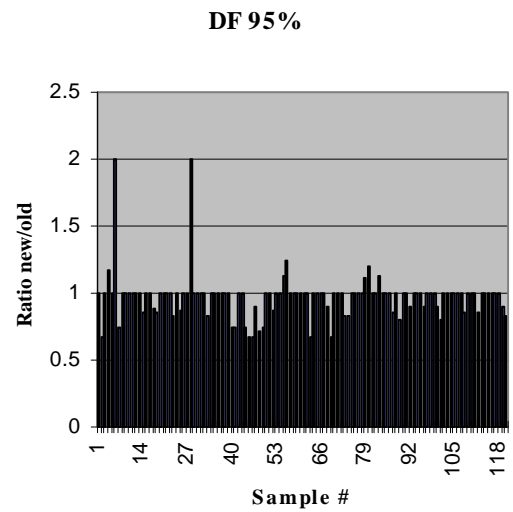


Figure 9.

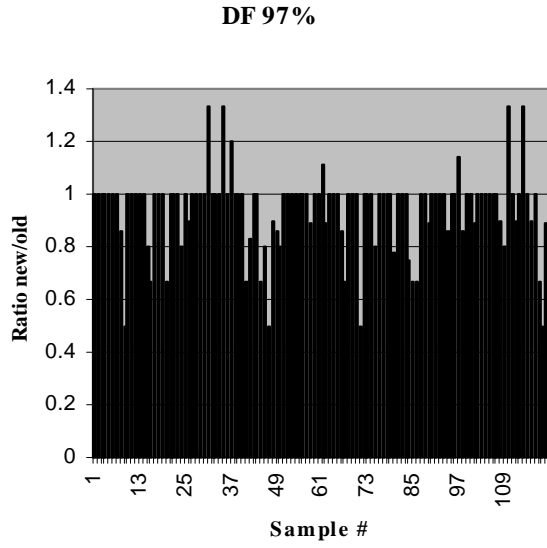


Figure 10.

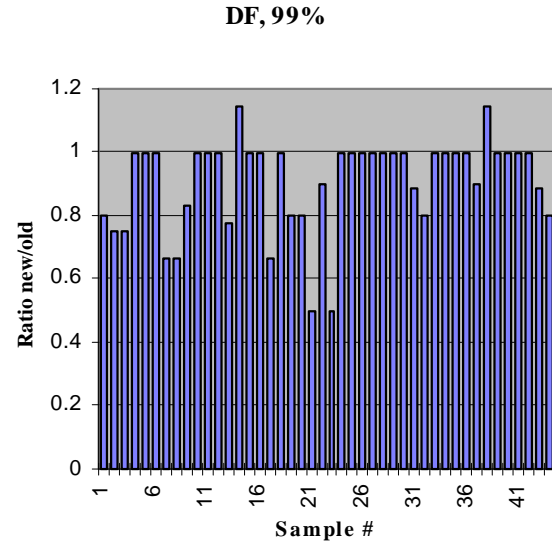


Figure 11.

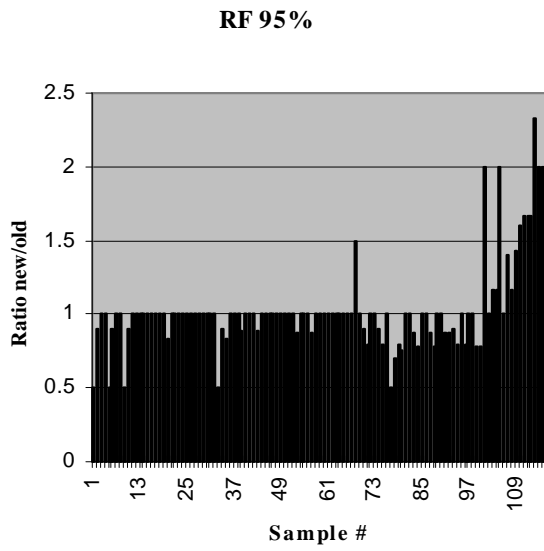


Figure 12.

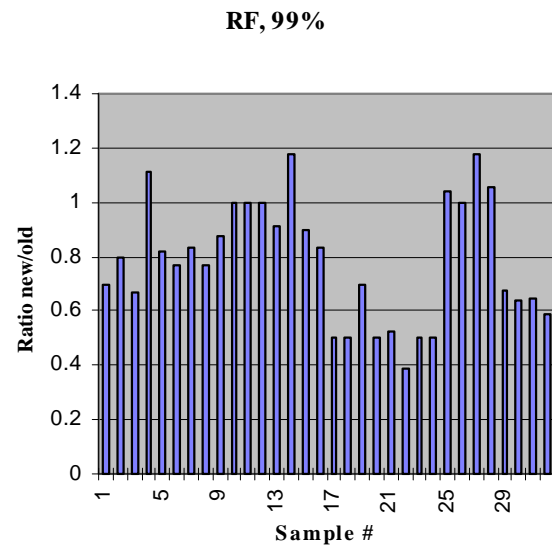


Figure 13.

Table 1. Cumulative frequencies for exposure factor parameters, with a comparison of data values (from the *Exposure Factors Handbook* tables cited in the text) and values predicted from the best fitting lognormal distributions used in the Monte Carlo analysis. This table is for variability of the ingestion rate per unit body mass for home produced beef, and corresponds to Figure 1. The values are EFH/log, where EFH (in the upper row) is the numerical value in the *Exposure Factors Handbook* and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value in the same row, as described in the text.

Cumulative Percentile									
Age Group (years)	1	5	10	25	50	75	90	95	99
6-11	0.17	0.31	0.36	0.62	1.00	2.10	5.43	5.95	6.33
	0.10	0.12	0.22	0.44	1.00	2.20	4.50	6.60	6.80
12-19	0.25	0.32	0.34	0.60	1.00	1.62	2.34	2.36	2.83
	0.25	0.30	0.40	0.60	1.00	1.60	2.50	3.10	3.20
Adult	0.17	0.22	0.25	0.43	1.00	1.72	3.07	4.09	5.19
	0.15	0.30	0.40	0.60	1.00	1.70	2.80	4.00	5.50

Table 2. Cumulative frequencies for exposure factor parameters, with a comparison of data values (from the *Exposure Factors Handbook* tables cited in the text) and values predicted from the best fitting lognormal distributions used in the Monte Carlo analysis. This table is for variability of the ingestion rate per unit body mass for home produced milk, and corresponds to Figure 2. The values are EFH/log, where EFH (in the upper row) is the numerical value in the *Exposure Factors Handbook* and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value for the measurements, as described in the text.

Cumulative Percentile									
Age Group (years)	1	5	10	25	50	75	90	95	99
All	0.03	0.06	0.07	0.29	1.00	1.87	2.38	3.01	3.56
	0.07	0.11	0.20	0.35	0.70	1.50	2.70	3.79	3.85

Table 3. Cumulative frequencies for exposure factor parameters, with a comparison of data values (from the *Exposure Factors Handbook* tables cited in the text) and values predicted from the best fitting lognormal distributions used in the Monte Carlo analysis. This table is for variability of the ingestion rate per unit body mass for recreational fishing, and corresponds to Figure 3. The values are EFH/log, where EFH (in the upper row) is the numerical value in the *Exposure Factors Handbook* and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value for the measurements, as described in the text.

Age Group (years)	Cumulative Percentile								
	1	5	10	25	50	75	90	95	99
All	NA*	NA	0.11	0.36	1.00	1.93	2.38	3.00	NA
	NA	NA	0.25	0.42	0.84	1.77	3.19	4.53	NA

NA* = Measurements not available at these percentiles

Table 4. Cumulative frequencies for exposure factor parameters, with a comparison of data values (from the *Exposure Factors Handbook* tables cited in the text) and values predicted from the best fitting lognormal distributions used in the Monte Carlo analysis. This table is for variability of the occupancy periods for farming populations (the only population for which this factor applies in the present analysis, and corresponds to Figure 4. The values are EFH/log, where EFH (in the upper row) is the numerical value in the *Exposure Factors Handbook* and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value in the same row, as described in the text.

Age Group (years)	Cumulative Percentile								
	1	5	10	25	50	75	90	95	99
All	NA	NA	NA	0.25	1.00	2.67	4.83	5.8	NA
	NA	NA	NA	0.50	1.00	2.20	4.60	7.00	NA

NA* = Measurements not available at these percentiles

Table 5. Cumulative frequencies for exposure factor parameters, with a comparison of data values (from the calculations reported in the text) and values predicted from the best fitting lognormal distributions used in the Monte Carlo analysis. This table is for variability of the age crossing correction factor, and corresponds to Figure 5. The values are EFH/log, where EFH (in the upper row) is the numerical value in the *Exposure Factors Handbook* and log (in the lower row) is the numerical value from the best-fitting lognormal distribution. All values in a row are the ratio of the percentile value over the median value for the measurements, as described in the text.

Age Group (years)	Cumulative Percentile								
	1	5	10	25	50	75	90	95	99
All	NA*	NA	0.82	0.92	1.00	1.20	1.30	1.45	NA
	NA	NA	0.80	0.90	1.00	1.13	1.25	1.30	NA

NA* = Simulations not available at these percentiles

Table 6. Summary of variability of exposure parameters for the three pathways and four age groups. All measures of variability are the geometric standard deviations associated with a lognormal distribution. All medians are 1.0 (including the median of the composite distribution for EF, given in the last column). The four entries are Ingestion Rate per Unit Body Mass (IR/BW); Occupancy Period or Exposure Duration (ED); Age Crossing Correction Factor (CF); and the Composite Geometric Standard Deviation (COMP). The mean for any of these distributions may be obtained from the formula (remembering that the median is 1.0): $\text{Mean} = \exp((\ln^2(\text{GSD}))/2)$

Receptor Population	IR	OP	ACF	COMP
Commercial Beef Farmer				
0-5 year age-group	3.3	3.2	1.2	5.3
6-11 year age-group	3.3	3.2	1.2	5.3
12-19 year age-group	2.0	3.2	1.2	3.9
>20 year age-group	2.3	3.2	1.0	4.2
Commercial Dairy Farmer				
0-5 year age-group	2.8	3.2	1.2	4.8
6-11 year age-group	2.8	3.2	1.2	4.8
12-19 year age-group	2.8	3.2	1.2	4.8
>20 year age-group	2.8	3.2	1.0	4.7
Recreational Fisher				
0-5 year age-group	2.8	NA*	NA	2.8
6-11 year age-group	2.8	NA	NA	2.8
12-19 year age-group	2.8	NA	NA	2.8
>20 year age-group	2.8	NA	NA	2.8

*NA=Not Applicable due to the factor being irrelevant for noncancer endpoints.

Table 7. Analysis of the fractional error (FE) introduced into the estimate of percentiles of the aggregated variability distribution for risk (or HI) as a result of numerical approximation of the sector-specific variability distribution PDFs.

N	f	FE (95th)	FE (97th)	FE (99th)
5	0.5	2.3*	2.4	2.5
	1	2.5	2.9	3.2
10	0.5	0.70	0.75	0.85
	1	0.82	0.90	0.95
20	0.5	0.12	0.14	0.17
	1	0.14	0.16	0.19
30	0.5	0.10	0.12	0.15
	1	0.12	0.13	0.17
40	0.5	0.09	0.10	0.12
	1	0.10	0.12	0.14

* a fractional error of 2.3 indicates a 230% error.

REFERENCES

- EPA's (U.S. Environmental Protection Agency). 1997. *Guiding Principles for Monte Carlo Analysis*.
- Morgan, M. and M. Henrion. 1990. *Uncertainty: A Guide to Dealing with Uncertainty in Quantitative Risk and Policy Analysis*. Cambridge University Press, pp. 88-90.
- Crawford-Brown, D. 1997. *Theoretical and Mathematical Foundations of Human Health Risk Analysis*. Kluwer Academic Press.
- EPA (U.S. Environmental Protection Agency). 1997. *Exposure Factors Handbook*.

Appendix J

Ecotoxicological Profile for Ecological Receptors

Ecotoxicological Profile for Selected Ecological Receptors 2,3,7,8-TCDD

This ecotoxicological profile on 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) contains five sections: (1) background, (2) geochemistry of the constituent in various ecological media, (3) effects characterization, (4) bioaccumulation potential, and (5) criteria development. The first four sections are intended to provide an overview of the environmental factors that influence the toxicological potential of TCDD so that the limitations of the criteria may be better understood. The fifth section presents the rationale and development of criteria for the suite of ecological receptors used to represent aquatic and terrestrial ecosystems. The profile is intended to present the ecotoxicological criteria in a broader environmental context, so the ecological significance of the criteria may be properly interpreted.

I. Introduction

The persistent, bioaccumulative, and hydrophobic nature of TCDD strongly influences this constituent's environmental behavior in ecological systems. Sediments and biota act as the primary sinks for TCDD. The movement of TCDD in the environment closely corresponds with sediment transport because TCDD is readily adsorbed to organic particulates. TCDD is also bioaccumulated through food chain mechanisms; however, evidence to support biomagnification has only been confirmed in aquatic food chains. The toxicity of TCDD has been narrowed to particularly sensitive vertebrate species, especially mammals. TCDD appears to exert toxicity through a receptor-specific mode of action, the aryl-hydrocarbon (Ah) receptor, that is found primarily in vertebrates. The characteristic dose-response curve of TCDD is steep with a narrow concentration range between no effects and lethal levels. Species particularly at risk include mammals and birds that could potentially have high TCDD exposure from consuming contaminated prey species (e.g., fish and invertebrates) (U.S. EPA, 1993a).

TCDD is commonly found in the environment as mixtures of polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDF), and dioxin-like polychlorinated biphenyls (PCBs). Multiple congeners within each constituent class have been measured in these mixtures. PCDDs have been identified as 75 congeners, PCDF as 135 congeners, and dioxin-like PCBs as 209 congeners. Assessing the potential for adverse toxic effects in ecosystems becomes increasingly difficult because congeners display varying toxicity and bioaccumulative potential. The approach implemented to assess toxicity and bioaccumulation of the mixture uses toxicity equivalency factors (TEFs) and bioaccumulative equivalency factors (BEFs). TEFs are adjustment factors that express the relative toxicity of each congener with respect to TCDD toxicity; BEFs determine the relative bioaccumulative potential congeners with respect to TCDD. When the mixture

composition is known and converted into TCDD equivalents, the sum of all toxicity equivalent concentrations, TEQCs, can be expressed additively as toxicity equivalents (TEqs). The TEQC for surface water, soil, or sediment can be compared directly to a 2,3,7,8- TCDD criteria to estimate the potential for adverse effects. The specific dioxin congeners are listed in Attachment 1.

II. Environmental Behavior of TCDD in Various Ecological Media

Overall, the movement of sediments, particulates, and soils via erosion closely mimics the mobility and fate of TCDD. For example, in surface water, TCDD is associated primarily with suspended organic matter which eventually settles into sediments. Concentrations in sediments range from 6.0E-05 to 7.6E-03 mg/kg sediment with the latter being related to sediments in areas of high industrial activity. In addition to the movement of TCDD via abiotic means, TCDD is also mobile through biotic means. Concentrations in fish range from below detection, 5.0E-07, to 1.0E-04 mg/kg fish tissue (whole body, wet weight). In a few cases, fish tissues have exceeded 1.0E-04 µg/kg fish tissue; however, this is rare. Over time, concentrations in sediment and biota decrease as TCDD is slowly metabolized or transported elsewhere through sediment movement. Similar chemical behavior is observed in terrestrial systems; however, TCDD is adsorbed to organic content in the soil and is somewhat less mobile (Eisler, 1986).

III. Effects Characterization

This section, along with the bioaccumulation potential section, is subdivided to evaluate receptors of the freshwater and terrestrial ecosystems separately. These sections summarize the range of effects data for receptors of concern.

Freshwater Ecosystems

Binding of TCDD to the Ah receptor has been identified as the primary mode of toxic action. Interestingly, this biochemical receptor appears to be present in some species and absent in others. In fish, adverse effects such as mortality, inhibited reproduction, and tissue damage have been noted during short-term exposures. In contrast, long-term studies exposing aquatic invertebrates (e.g., snails, worms, daphnids) and aquatic plants indicate no adverse toxic effects. Vertebrate species have characteristically indicated higher sensitivity to TCDD exposures than invertebrates. The difference between vertebrate and invertebrate toxicity ranges can be as high as four orders of magnitude. Several studies indicate that no adverse effects are suggested at TCDD concentrations of 1.3 µg TCDD/L for freshwater species of algae, aquatic plants, and invertebrates. In sharp contrast, experiments exposing early lifestages of fish to TCDD have reported LC₅₀s as low as 0.000046 µg TCDD/L (U.S. EPA, 1993a).

A large amount of ecotoxicity data has been generated for fish species because of their sensitivity to TCDD exposures. Interestingly, toxicity tests indicate latent threshold responses of species exposed to TCDD. When this occurs, a typical toxicity test indicates no observable adverse effects during exposures, but, up to 100 days after exposure to TCDD, lethality within the test group is observed. For example, observations of juvenile salmon following acute 4-day exposures to 5.6 ng TCDD/L indicated minimal effects; however, these same test species showed 100% lethality 56 days after exposures had ceased. To contrast, similar experiments exposing juvenile salmon to concentrations of 0.56 ng TCDD/L indicated no adverse effects during both

the 4-day exposure period and the extended observational period after exposure (i.e., 114 days). These studies indicate the unpredictability of latent lethal effects in fish species. Chronic exposure durations of 21 days reported an LC_{50} in rainbow trout sac fry at 1.8 ng TCDD/L. Rainbow trout swim-up fry appeared more sensitive to TCDD exposure with a NOEC of 0.001 ng/L and an LC_{50} of 0.046 ng/L. Adverse effects to reproductive, developmental, and growth endpoints during chronic exposures of variable length are noted in juvenile fish from 0.038 to 0.1 ng/L. The trend in TCDD toxicity to fish species is that the concentration range between no effects, low effects, and lethality is relatively narrow, indicating a steep dose-response curve. Further, latent effects from acute exposure can result in unexpected high mortality within fish populations. Given this finding, a conservative approach for deriving protective concentration for vertebrate species appears appropriate (U.S. EPA, 1993a). Ecotoxicity data reporting effects levels for freshwater invertebrates indicate that these receptors are relatively tolerant to TCDD exposures. No effects to reproduction or growth were evident in annelids (*Paranais sp.*), molluscs (*Physa sp.*), and arthropods (*Aedes aegypti*) at 200 ng TCDD/L.

The ecotoxicity database at higher trophic levels of aquatic ecosystems indicated elevated sensitivity in species of mammals and birds also. Studies characterizing the toxicity of TCDD to mink indicated adverse effects to receptors at single doses of 4,200 pg/g body weight with latent mortality occurring 28 days after exposures have ceased. Avian receptors that prey within the aquatic ecosystem, such as the mallard, appear less sensitive than the mink. A single ingestion dose to mallards resulted in an LD_{50} of 15,000 pg TCDD/g body weight after 37 days of observation.

Terrestrial Ecosystems

Terrestrial vertebrate receptors portray similar sensitivities to TCDD exposure. Acute mammalian toxicity occurs in exposure ranges of approximately 2 to 115 μ g TCDD/kg body weight following single-dose exposures (Eisler, 1986). In avian receptors characteristic of the terrestrial receptors, lethality (LC_{50} s) ranges from approximately 15 to greater than 810 μ g/kg body weight with the Northern bobwhite quail and the domestic chicken being the more sensitive receptors. Ovo injections to developing ring-necked pheasants indicated LD_{50} s of 1,354 to 2,182 pg TCDD/g egg (Nosek et al., 1993). Chronic exposures of 0.001 μ g/kg-d to rats indicated no effects to reproductive endpoints after a 2-year duration. Other terrestrial receptors such as plants and earthworms do not indicate sensitivity to TCDD exposure.

IV. Bioaccumulation Potential

A second issue related to the overall impact of TCDD is its ability to be transferred through the food chain to higher trophic levels. Given the lipophilic nature of TCDD, the potential for bioaccumulation and biomagnification is high. TCDD is quickly mobilized into fat tissue where it is minimally metabolized and eliminated. Since vertebrates predominate at higher trophic levels and these receptors appear to be highly sensitive, TCDD poses a significant threat to upper trophic level predators. This section presents biological uptake measures (e.g., BCFs, and BAFs) used to derive protective surface water and soil concentrations for constituents considered to bioconcentrate and/or bioaccumulate in the generic aquatic ecosystems.

Freshwater Ecosystems

Bioaccumulation of TCDD has been widely observed in freshwater systems. The BCFs for algae estimated from tissue residue studies ranged from 300 to 1,200 after 32-day exposures. Duckweed (*Lemna sp.*), a submersed macrophyte, bioconcentrates TCDD also with BCFs reported at approximately 4,000. Bioconcentration has also been indicated in species of aquatic invertebrates. Initially, BCFs were reported at 380 and 1,200 for snails and daphnids, respectively; however, upon exposing the same receptors to lower concentrations of TCDD, higher BCFs were indicated. Following lower exposures, snails indicated BCFs ranging from 1,040 to 2,310 whereas daphnids indicated BCFs from 2,830 to 4,070 (U.S. EPA, 1993a). Early life stage tests of amphibians have also indicated bioconcentration; however, the magnitude is not as high as for other freshwater receptors. Amphibian eggs and tadpoles exposed to TCDD have indicated that bioconcentration factors range from 0.6 to 4 for eggs and 0.7 to 19 for tadpoles. The lower potential for bioaccumulation is probably related to the high elimination rates of TCDD in amphibian species. Once amphibians are removed from surface water contaminated with TCDD, the half-life of TCDD in tissues can range from 1 to 7 days (Jung and Walker, 1997).

Bioaccumulation of TCDD in fish has been extensively documented. The discussion of TCDD bioaccumulation in fish will consistently present bioaccumulation factors on a lipid basis since TCDD is predominantly stored in the adipose tissue of organisms. Bioconcentration factors for fish species range from 81,300 to 4,300,000 L/kg based on surface water exposures (U.S. EPA, 1993a). Continued research suggests that BCFs and BAFs based on surface water concentrations may not be reliable because of interfering substances and minimum detection level in measurement. For extremely hydrophobic constituents, such as TCDD, EPA has stated that reliable measurements of ambient water concentrations (especially dissolved concentrations) are not available for TCDD and that accumulation of these constituents in fish or other aquatic organisms cannot be referenced to a water concentration as required for a BCF or BAF (U.S. EPA, 1993a). Problems in calculating a BCF/BAF occur because TCDD levels below detection can also bioaccumulate in fish even though concentrations of TCDD in the water column cannot be measured. Given these limitations, the accuracy of TCDD measurement and BAF estimation using surface water concentrations may misrepresent actual bioaccumulation. Fortunately, extremely hydrophobic constituents can be measured in sediments and aquatic life and, because these chemicals tend to partition to lipids and organic carbon, a biological uptake factor that reflects the relationship between sediment concentrations and organism concentrations may be more appropriate. Consequently, the biota-sediment accumulation factor (BSAFs) is the preferred metric for accumulation for extremely hydrophobic chemicals (e.g., chemicals with $\geq \log K_{ow}$ of ~ 6.5). Concentrations in sediment are more readily measured at detectable levels and can be used to determine BSAFs in freshwater species. BSAFs in fish range from 0.03 to 0.2 kg sediment/kg tissue while invertebrate species indicate BSAFs of 0.48 kg sediment/kg tissue for the sandworm, 0.73 kg sediment/kg tissue for shrimp, and 0.93 for clams (U.S. EPA, 1993a). When partitioning of constituents between sediment particles, pore water, and surface water are accounted for, good correlation between BSAFs and surface-water-derived BAFs is noted.

In freshwater ecosystems, the method used to assess exposure of TCDD to receptors further up the food chain applied BSAFs. Several sources were identified to derive BSAF values representative of fish across the nation. BSAFs in [mg congener/kg LP]/[mg congener/kg

sediment OC] were calculated from measured data identified in the references. A description of the database, scope, and methodology implemented in each report is outlined under each reference.

- # *Interim Report on Data and Methods for Assessment of 2,3,7,8- Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife* (U.S. EPA, 1993a)

This report calculated BSAFs for TCDD based on measured TCDD residues in fish tissues and estimated TCDD sediment concentrations collected in Lake Ontario (U.S. EPA, 1990 as cited in U.S. EPA, 1993a). Five fish species were sampled over a 2-year period. In this study, BSAFs were derived only for TCDD residues in trophic levels 2, 3, and 4 of aquatic receptors. When this report was published, U.S. EPA (1993a) data were the most comprehensive study data available. This study reported that BSAFs varied depending on the age of the species, the primary habitat (i.e., near shore, deep water), and feeding preferences of fish species. Because of the continued loading of TCDD compounds to Lake Ontario, the sediment, surface water, and fish tissue concentrations were not predicted to be at equilibrium. This disequilibrium may underpredict BSAF values. Conversations with Phil Cooke confirmed this uncertainty.

- # *Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors* (U.S. EPA, 1995a).

This document utilized the same data set from the Lake Ontario study for TCDD BSAF derivation along with data collected by Niimi and Oliver (1989). Additional analysis of original samples was undertaken to measure concentrations of other PCDD, PCDF, and PCB in sediments and fish tissues. These data were added to the analysis to determine trophic level 3 BSAFs for fish of multiple dioxin and furan congeners. Because these data were derived from the same sample pool the uncertainties regarding disequilibrium remain.

- # CT DEP (Connecticut Department of Environmental Protection). 1992. *Report to the Connecticut General Assembly Department Summary of the Dioxin Monitoring program 1987 to 1991*. Connecticut Department of Environmental Protection.

Bauer, K. 1992. *Multivariate Statistical Analyses of Dioxin and Furan Levels in Fish, Sediment, and Soil Samples Collected Near Resource Recovery Facilities*. Final Report. Compiled for Connecticut Department of Environmental Protection, Water Compliance Unit. Prepared by Midwest Research Institute. December.

These documents reported measurements of dioxin and furan congeners in sediment and fish tissue collected in waterbodies in the vicinity of five resource recovery facilities (RRFs). The results were assessed by the State of Connecticut's Department of Environmental Protection to assess whether emissions from RRFs may influence background concentrations of dioxin compounds in ecological receptors and media. Residues of PCDDs and PCDFs were measured in fish, sediment, and soil. Samples were collected over a 4-year period between sites that were grouped as control, preoperational, and operational. The primary objective of the study was to determine whether RRF

incineration would influence PCDD and PCDF levels. The important elements reported in the study were obviously the fish tissue concentrations and the sediment concentrations. The issue of equilibrium is also in question in this study due to the continued loading of dioxin congeners to the waterbodies from facilities operation during the study. However, because the duration of sampling was longer, a greater likelihood of observing levels closer to equilibrium is indicated.

The State of Connecticut values were used to determine food chain exposures to receptors that forage in the freshwater ecosystem. Calculated BSAF values are provided in Section 5.4.1.6. These data were more comprehensive, reporting over 200 fish tissue concentrations in six species and over 150 sediment concentrations for both preoperational and operational sites. Samples were collected from multiple waterbodies surrounding each facility over a 4-year period. Further, the variability in fish lipid content and sediment organic content was characterized by 550 and 343 samples, respectively. Unfortunately, the report pooled the data across sites rather than reporting on a site-specific basis. This prohibited the relative comparison across sites.

Terrestrial Ecosystem

Terrestrial receptors also indicate levels of bioaccumulation; however, data are somewhat limited to adequately characterize the potential for bioaccumulation in mammalian receptors. Bioaccumulation factors and in small mammals have been measured from 0.3 to 7.2 (Sample et al., 1998a). The BCFs in insects and other invertebrates of 1.3 have been reported (Sample et al., 1998b; Abt and Associates, 1993). One specific group of invertebrates, earthworms, which have been more extensively investigated, accumulates TCDD in the range of 5 to 22 kg soil/kg tissue (Sample et al., 1998b). The bioconcentration of TCDD in plant tissues has not been sufficiently characterized. Because TCDD has been associated with acute toxicity, further investigations are required before sufficient data are available to characterize this source of exposure to consuming terrestrial organisms especially herbivores.

V. Criteria Development

Mammals: Murray et al. (1979) exposed three generations of Sprague-Dawley rats to diets containing 0, 0.001, 0.01, or 0.1 µg TCDD/kg-day. At the 0.01 µg/kg-day dose, Murray et al. (1979) observed no effect on fertility among the f_0 rats, but a significant reduction in fertility was observed among the f_1 and f_2 rats. Thus, through three successive generations, the reproductive capacity of rats ingesting TCDD was clearly affected at dose levels of 0.01 and 0.1 µg/kg-day, but not at 0.001 µg/kg-day. This study was selected for benchmark derivation because it consists of a multigenerational exposure scenario that demonstrates a clear dose-response for reproductive effects attributable to TCDD.

Other studies that were considered for benchmark identification include one subchronic study documenting TCDD exposure to mammalian wildlife species. Hochstein et al. (1988) administered TCDD dietary concentrations of 0, 0.001, 0.01, 0.1, 1.0, 10, and 100 ppb to mink for 125 days. While no significant adverse effects were observed on mink fed dietary concentrations of 0.1 ppb or less, mortality was noted in groups fed 1 and 10 ppb. Several studies have documented subchronic and chronic exposure of TCDD to laboratory animals. Khera and Ruddick (1972) (as cited in U.S. EPA, 1995b) assessed the postnatal effect of TCDD on pregnant

Wistar rats. In this experiment, rats were given 0, 0.125, 0.25, 0.5, or 1.0 µg TCDD/kg-day from days 6 through 15 of gestation. Dose-related decreases in the average litter size and pup weight at birth were noted in all but the 0.125-µg/kg-day dose. Bowman et al. (1989a, 1989b) studied the reproductive effects of Rhesus monkeys exposed to diets containing 5 ppt and 25 ppt TCDD for 7 and 24 months. The female monkeys exposed to 25 ppt had a significantly lower Index of Overall Reproductive Success (IORS), while the 5-ppt group did not differ from the control. The 5 ppt was converted to a dose of 0.00013 µg/kg-day using the study's daily allotment of 200 grams of monkey feed and the typical female monkey's body weight outlined in *Recommendations for and Documentation of Biological Values for Use in Risk Assessment* (U.S. EPA, 1988).

The 125-day test performed by Hochstein et al. (1988) was not considered as appropriate for deriving a benchmark since the study was subchronic rather than chronic and the perceived endpoints focus more on mortality than reproductive effects. The Murray et al. (1979) study was chosen over the Khera and Ruddick study (as cited in U.S. EPA, 1995b) because of a lower reported NOAEL for rats. The reproduction study by Bowman et al. (1989a, 1989b) on Rhesus monkeys (which produced a lower NOAEL) was not selected because the Murray et al. (1979) study incorporated a multigenerational exposure regime and contained stronger dose-response information.

The NOAEL of 1.0E-6 mg/kg-d from the Murray et al. (1979) was scaled for species representative of freshwater and terrestrial ecosystems using a cross-species scaling algorithm adapted from Sample et al. (1997)

$$Benchmark_w = NOAEL_t \times \left(\frac{BW_t}{BW_w} \right)^{1/4}$$

where $NOAEL_t$ is the NOAEL (or LOAEL/10) for the test species, BW_w is the body weight of the wildlife species, and BW_t is the body weight of the test species. This is the default methodology EPA proposed for carcinogenicity assessments and reportable quantity documents for adjusting animal data to an equivalent human dose (57FR 24152). Since the Murray et al. (1979) study documented reproductive effects from TCDD exposure to three generations of male and female rats, the mean male and female body weight for each representative species was used in the scaling algorithm to obtain the toxicological benchmarks.

Data were available on the reproductive, developmental, and growth effects of TCDD. In addition, the data set contained studies that were conducted over chronic and subchronic durations and during sensitive life stages. Most of the studies identified were conducted using laboratory mammals and, as such, interspecies differences among wildlife were not identifiable. Therefore, the data set does not support an uncertainty factor to account for interspecies differences in toxicological sensitivity. The reproductive NOAEL selected from Murray et al. (1979) was within an order of magnitude of the lowest identified NEL or LEL, and, therefore, the benchmarks developed for mammals representative of freshwater and terrestrial ecosystem were categorized as **adequate**.

Birds: In many field studies, reduced reproduction levels in avian species have been correlated to TCDD equivalents; however, the dose-response relationship specific to TCDD itself cannot be determined from the effects of other contaminants. The only identified research investigating the subchronic toxicity of TCDD among avian species was performed by Nosek et al. (1992). Ring-necked pheasants were dosed weekly for 10 weeks by ip injection at an equivalent rate of 0.14, 0.014, and 0.0014 µg TCDD/kg-day (weekly dose was divided by 7 for the equivalent daily dose). Cumulative egg production was significantly reduced among pheasants exposed to 0.14 µg TCDD/kg-day, but not among those pheasants exposed to the two lower doses.

The pheasant reproductive effect NOAEL of 0.014 µg/kg-day for TCDD (Nosek et al., 1992) was used in calculating avian wildlife benchmarks. The Nosek et al. (1992) study demonstrates a clear dose-response to a critical reproductive endpoint and is based on an exposure lasting more than 28 days. This study should be interpreted judiciously since it involves an ip injection rather than an oral route of administration. Assuming 100% absorption from ip injection, the ip exposure route may overestimate the absorption rate of TCDD via oral ingestion by a factor of 1 to 5 depending upon diet composition (Abt & Associates, 1993).

The principles for allometric scaling were assumed to apply to birds, although specific studies supporting allometric scaling for avian species were not identified. Thus, for avian species representative of a freshwater ecosystem, the NOAEL of 0.014 µg/kg-d from Nosek et al. (1992) was scaled using the cross-species scaling method of Sample et al. (1997). Since the Nosek et al. (1992) study documented reproductive effects from TCDD exposure to female pheasants, female body weights for each representative species were used in the scaling algorithm to obtain the toxicological benchmarks. Although there is no formal designation for benchmarks developed from ip exposure route studies, the benchmarks derived from Nosek et al. (1992) were categorized as **interim** based on the absorption uncertainties surrounding the intraperitoneal injection of TCDD to pheasants.

Freshwater Community: Since an AWQC for TCDD was not available and a Secondary Chronic Value (SCV) could not be calculated because of limited ecotoxicity data, a benchmark protective of the freshwater community was not established. Numerous fish studies documenting the effects of chronic TCDD exposure were identified. The rainbow trout is one of the most extensively studied aquatic organisms for effects from TCDD exposure. The lowest identified toxicity values for TCDD exposure to rainbow trout were a 4-day LC₅₀ of 1.83 ng/L (U.S. EPA, 1993a) and a LOAEL of 0.038 ng/L based on 45% mortality during a 28-day exposure (Mehrle et al., 1988). Based on the current data set, TCDD appears highly toxic to aquatic organisms. This concern has prompted further research to characterize the toxicity of TCDD in fish and aquatic invertebrates species creating a database sufficient to calculate a Final Chronic Value (FCV) or SCV. A narrative criteria of 0.6 pg/L in surface water has been proposed for fish. This concentration approximates a level at which the potential for adverse effects is low for most fish species. Adverse effects to sensitive fish species are indicated at 1.0 pg/L (U.S. EPA, 1993a).

Algae and Aquatic Plants: The toxicological benchmarks for aquatic plants were either: (1) a no observed effects concentration (NOEC) or a lowest observed effects concentration (LOEC) for vascular aquatic plants (e.g., duckweed) or (2) an effective concentration (EC_{xx}) for a species of freshwater algae, frequently a species of green algae (e.g., *Selenastrum*

capricornutum). Aquatic plant data were not identified for TCDD and, therefore, no benchmark was developed.

Benthic Community: Benchmarks for the protection of benthic organisms were determined using the Equilibrium Partition (EQ_p) method. The EQ_p method uses a Final Chronic Value (FCV) or other chronic water quality measure, along with the fraction of organic carbon and the octanol-carbon partition coefficient (K_{oc}) to determine a chemical concentration that may be present in the sediment while still protecting the benthic community (U.S. EPA, 1993b). The EQ_p number is the best recommendation of a chemical concentration that may be present in the sediment while still protecting the benthic community from harmful effects resulting from possible chemical exposure. Since there is no AWQC, FCV, or SCV, the benchmark for the benthic community was not calculated for TCDD. Preliminary screening values currently being reviewed include the sediment TRV proposed by EPA Region 6 of 0.032 μg 2,3,7,8-substituted dioxin congeners/kg sediment (dry weight). In comparison, recent studies have indicated no adverse effects to amphipods exposed for 10 days to 25 μg TCDD/kg sediment (Barber et al., 1998). Overall these results support previous findings that benthic invertebrates are not acutely sensitive to 2,3,7,8- TCDD exposure. The general consensus among researchers is that insufficient toxicological data are available to develop a community-based sediment criteria for TCDD.

Terrestrial plants: Adverse effects levels for terrestrial plants were identified for endpoints ranging from percent yield to root length. As presented in Efroymson et al. (1997), phytotoxicity benchmarks, were selected by rank-ordering the LOEC values and then approximating the 10th percentile. If there were 10 or fewer values for a chemical, the lowest LOEC was used. If there were more than 10 values, the 10th percentile LOEC was used. Such LOECs applied to reductions in plant growth, yield reductions, or other effects reasonably assumed to impair the ability of a plant population to sustain itself, such as a reduction in seed elongation. However, terrestrial plant studies were not identified for TCDD and, as a result, a benchmark could not be developed.

Soil Community: Adequate data with which to derive a benchmark protective of the soil community were not identified.

Attachment 1. List of Furan and Dioxin Congeners of Ecological Concern

Furans

1, 2, 3, 4, 6, 7, 8-Heptachlorodibenzofuran (1, 2, 3, 4, 6, 7, 8-HpCDF)
1, 2, 3, 4, 7, 8, 9-Heptachlorodibenzofuran (1, 2, 3, 4, 7, 8, 9-HpCDF)
1, 2, 3, 7, 8, 9-Hexachlorodibenzofuran (1, 2, 3, 7, 8, 9-HxCDF)
1,2, 3, 4, 7, 8-Hexachlorodibenzofuran (1,2, 3, 4, 7, 8-HxCDF)
1, 2, 3, 6, 7, 8-Hexachlorodibenzofuran (1, 2, 3, 6, 7, 8-HxCDF)
2, 3, 4, 6, 7, 8-Hexachlorodibenzofuran (2, 3, 4, 6, 7, 8-HxCDF)
1, 2, 3, 7, 8-Pentachlorodibenzofuran (1, 2, 3, 7, 8-PeCDF)
2, 3, 4, 7, 8-Pentachlorodibenzofuran (2, 3, 4, 7, 8-PeCDF)
2, 3, 7, 8-Tetrachlorodibenzofuran (2, 3, 7, 8-TCDF)
Octochlorodibenzofuran (OCDF)

Dioxins

1,2, 3, 4, 6, 7,8- Heptachlorodibenzodioxin (1, 2, 3, 4, 6, 7,8- HpCDD)
1, 2, 3, 4, 7, 8-Hexachlorodibenzodioxin (1, 2, 3, 4, 7, 8-HxCDD)
1, 2, 3, 6, 7, 8-Hexachlorodibenzodioxin (1, 2, 3, 6, 7, 8-HxCDD)
1, 2, 3, 7, 8, 9-Hexachlorodibenzodioxin (1, 2, 3, 7, 8, 9-HxCDD)
1, 2, 3, 7, 8-Pentachlorodibenzodioxin (1, 2, 3, 7, 8-PeCDD)
2, 3, 7, 8-Tetrachlorodibenzodioxin (2, 3, 7, 8-TCDD)
Octochlorodibenzodioxin (OCDD)

References

- Abt (Abt Associates, Inc.). 1993. *Revision of Assessment of Risks to Terrestrial Wildlife from TCDD and TCDF in Pulp and Paper Sludge*. Prepared for U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics, by Abt Associates, Inc., Bethesda, MD.
- Barber, T.R., D.J. Chappie, D.J. Duda, P.C. Fuchsman, and B.L. Finley. 1998. Using a spiked sediment bioassay to establish a no-effect concentration for dioxin exposure to the amphipod *Ampelisca abdita*. *Environmental Toxicology and Chemistry* 17(3):420-424.
- Bauer, K.M. 1992. *Multivariate Statistical Analyses of Dioxin and Furan Levels in Fish, Sediment, and Soil Samples Collected Near Resource Recovery Facilities. Final Report*. Prepared for Connecticut Department of Environmental Protection. Midwest Research Institute, Kansas City, MO.
- Bowman, R.E., S.L. Schantz, M.L. Gross, and S.A. Ferguson. 1989a. Behavioral effects in monkeys exposed to 2,3,7,8-TCDD transmitted maternally during gestation and four months of nursing. *Chemosphere* 18(1-6):235-242.
- Bowman, R.E., S.L. Schantz, N.C.A. Weerasinghe, M.L. Gross, and D.A. Barsotti. 1989b. Chronic dietary intake of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) at 5 or 25 parts per trillion in the monkey: TCDD kinetics and dose-effect estimate of reproductive toxicity. *Chemosphere* 18(1-6):243-252.
- CT DEP (Connecticut Department of Environmental Protection). 1992. *Report to the Connecticut General Assembly Department Summary of the Dioxin Monitoring Program 1987 to 1991*. As cited in Bauer, 1992.
- Efroymson, R.A., M.E. Will, G.W. Suter, II, and A.C. Wooten. 1997. *Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision*. ES/ER/TM-85/R3. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Lockheed Martin Energy Systems, Inc., Oak Ridge, TN.
- Eisler, R. 1986. Dioxin hazards to fish, wildlife, and invertebrates: a synoptic review. In: *Contaminant Hazard Reviews, Report No. 8*. U.S. Fish and Wildlife Service, U.S. Department of the Interior, Laurel, MD.
- Hochstein, J.R., R.J. Aulerich, and S.J. Bursian. 1988. Acute toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin to mink. *Archives of Environmental Contamination and Toxicology* 17:33-37.
- Jung, R.E., and M.K. Walker. 1997. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on development of anuran amphibians. *Environmental Toxicology and Chemistry* 16(2):230-240.

- Khera, K.S., and J.A. Ruddick. 1973. Polychlorodibenz-*p*-dioxins: perinatal effects and the dominant lethal test in wistar rats. In: *Chlorodioxins - Origin and Fate. A Symposium Sponsored by the Division of Pesticide Chemistry at the 162nd Meeting of the American Chemical Society*, E.H. Blair (ed.), 8th Edition. pp. 70-84. American Chemical Society, Washington, DC. September 16-17, 1971.
- Mehrle, P.M., D.R. Buckler, E.E. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. De Graeve, J.J. Coyle, and W.J. Adams. 1988. Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzodioxin and 2,3,7,8-tetrachlorodibenzofuran in rainbow trout. *Environmental Toxicology and Chemistry* 7:47-62.
- Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A. Schwetz. 1979. Three-generation reproduction study of rats given 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in the diet. *Toxicology and Applied Pharmacology* 50:241-252.
- Niimi, A.J., and B.G. Oliver. 1989. Assessment of the relative toxicity of chlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in Lake Ontario salmonids to mammalian systems using toxic equivalent factors (TEFs). *Chemosphere* 18(7/8):1413-1423.
- Nosek, J.A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. Toxicity and reproductive effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in ring-necked pheasant hens. *Journal of Toxicology and Environmental Health* 35:187-198.
- Nosek, J.A., J.R. Sullivan, S.R. Craven, A. Gendron-Fitzpatrick, and R.E. Peterson. 1993. Embryotoxocity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in the ring-necked pheasant. *Environmental Toxicology and Chemistry* 12:1215-1222.
- Sample, B.E., M.S. Aplin, R.A. Efroymson, G.W. Suter, II, and C.J.E. Welsh. 1997. *Methods and Tools for Estimation of the Exposure of Terrestrial Wildlife to Contaminants*. ORNL/TM-13391. Prepared for Office of Environmental Policy and Assistance, U.S. Department of Energy. Prepared by Oak Ridge National Laboratory, Oak Ridge, TN.
- Sample, B.E., J.J. Beauchamp, R.A. Efroymson, and G.W. Suter, II. 1998a. *Development and Validation of Bioaccumulation Models for Small Mammals*. ES/ER/TM-219. Prepared for Office of Environmental Management, U.S. Department of Energy. Oak Ridge National Laboratory, Oak Ridge, TN.
- Sample, B.E., J.J. Beauchamp, R.A. Efroymson, G.W. Suter, II, and T.L. Ashwood. 1998b. *Development and Validation of Bioaccumulation Models for Earthworms*. ES/ER/TM-220. Prepared for Office of Environmental Management, U.S. Department of Energy. Oak Ridge National Laboratory, Oak Ridge, TN.
- U.S. EPA (Environmental Protection Agency). 1988. *Recommendations for and Documentation of Biological Values for Use in Risk Assessment*. EPA/600/6-87/008. Office of Health and Environmental Assessment, Cincinnati, OH.

- U.S. EPA (Environmental Protection Agency). 1990. *Assessment of Risks from Exposure of Humans, Terrestrial, and Avian Wildlife, and Aquatic Life to Dioxins and Furans from Disposal and Use of Sludge from Bleached Kraft and Sulfite Pulp and Paper Mills*. EPA-560-/5-90-013. Prepared by Abt Associates, Inc., for the Office of Pesticides and Toxic Substances, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1993a. *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife*. EPA/600/R-93/055. Office of Research and Development, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1993b. *Technical Basis for Deriving Sediment Quality Criteria for Nonionic Organic Contaminants for the Protection of Benthic Organisms by Using Equilibrium Partitioning*. EPA-822-R-93-011. Office of Water, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1995a. *Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors*. EPA-820-B-95-005. Office of Water, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1995b. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,7,8-TCDD, and PCBs*. EPA-820-B-95-008. Office of Water, Washington, DC.

Ecotoxicological Profile for Ecological Receptors

Lead

This ecotoxicological profile on lead contains five sections: (1) background (e.g., background concentrations), (2) geochemistry of the constituent in various ecological media, (3) effects characterization, (4) bioaccumulation potential, and (5) criteria development. The first four sections are intended to provide an overview of the environmental factors that influence the toxicological potential of lead so that the limitations of the criteria may be better understood. The fifth section presents the rationale and development of criteria for the suite of ecological receptors used to represent aquatic and terrestrial ecosystems. This profile is intended to present the ecotoxicological criteria in a broader environmental context, so the ecological significance of the criteria may be properly interpreted.

I. Background

Lead is a nonessential, highly toxic heavy metal for which all known effects on biological systems are deleterious. Lead is present in low concentrations throughout the environment as a result of geologic weathering, with an average abundance in the earth's crust of 16 ppm. Human activities have resulted in a widespread increase in lead residues in the environment. In soils, natural background concentrations are generally on the order of 10 to 30 ppm, but, near lead emissions sources such as roadways, concentrations of up to 2,000 ppm have been found.

Naturally occurring lead has three oxidation states: elemental (0), divalent (+2), and tetravalent (+4). In its inorganic forms, lead is found primarily in the divalent state. Organolead compounds, the most important of which are tetramethyl and tetraethyl lead, are formed predominantly by lead in the tetravalent state, and are considered to be the more toxic forms. In most of its forms, except for some lead salts, lead is relatively insoluble in water and tends to accumulate in sediments. The majority of lead ingested by biota is rapidly egested (Eisler, 1988). Inhaled lead, though, is absorbed quickly by blood (ATSDR, 1997). Lead does bioconcentrate, and older organisms tend to have the highest body burdens. Biomagnification of lead in the food chain, though, has been found to be negligible (Eisler, 1988).

II. Geochemistry of Lead in Various Ecological Media

Lead in Soils

The speciation of lead in soils is dependent on physicochemical processes including adsorption, precipitation, and complexation with solid and aqueous inorganic and organic phases within the soil. These processes are themselves determined by such factors as soil pH, organic matter concentrations, lead concentrations, and the presence of other inorganic components (NSF, 1977, cited in ASTDR, 1997). The atmospheric deposition rate for lead is the primary factor defining its accumulation in most soils (ASTDR, 1997).

- # Lead speciation in soils is dependent on physico-chemical processes including adsorption, precipitation, and complexation.
- # Most lead in soils is strongly sorbed to organic matter and very little is transported to surface or groundwater.

Most of the lead in soils is strongly sorbed to organic matter and very little is transported to surface water or ground water (ASTDR, 1997). Ion exchange processes with hydrous oxides or clays, or chelation with humic or fulvic acids can remove lead from solution in soil (Olson and Skogerboe, 1975, cited in ASTDR, 1997). In soils with pH ≥ 5 and $\geq 5\%$ organic matter content, atmospheric lead is retained within the uppermost 2-5 cm of undisturbed soil (ASTDR, 1997). In soils with pH 6-8 and a high organic matter content, lead can form insoluble organo-complexes. Within the same pH range but with a lower organic matter content, hydrous lead oxide complexes may form or lead may precipitate out with carbonate or phosphate ions (ASTDR, 1997). At lower pHs of 4-6 organo-lead complexes may be soluble (U.S. EPA, 1986, cited in ASTDR, 1997).

Lead in Surface Waters

A review of trace elements in rivers by Hart and Hines (1995) tabulated typical dissolved (i.e., $<0.4 \mu\text{m}$) lead concentrations ranging from 87 to 1,800 ng/l. The behavior of lead in rivers is primarily controlled by the balance between complexation with dissolved organic matter and association with suspended particulate matter (SPM) and colloidal matter (Hart and Hines, 1995).

Particles settling through surface waters can control the behavior of elements such as lead that are removed from the dissolved phase (usually $< 0.4 \mu\text{m}$) by forming nuclide/particle surface site complexes (Santschi, 1988 and references therein). Reactions with dissolved and particulate organic carbon can also regulate the concentration of organically complexed elements such as lead. These reactions can be particularly important in coastal waters that have high organic loadings and in estuarine environments that have large ionic strength gradients (Santschi, 1988).

- # The behavior of lead is primarily controlled by the balance between complexation with dissolved organic matter and association with SPM and colloids.
- # In a study of three U.S. rivers, lead was found to be partitioned between particulate, colloidal, and “truly” dissolved phases. Partitioning between filter-retained and filtrate lead showed a dependence on the concentration of total SPM.

Benoit (1995) determined lead concentrations in fresh water from three rivers in the northeast United States and investigated the relationship between lead in particulate, colloidal, and “truly” dissolved (i.e., occurring as individual solvated ions) phases. Partitioning between (0.45 μm) filter-retained and filtrate ($< 0.45 \mu\text{m}$) fractions exhibited a dependence on the concentration of total suspended solids (Benoit, 1995). This phenomenon, called the particle concentration effect, can be explained by the contribution of lead bound to colloids, which are included in the filter-passing fraction of conventionally “dissolved” trace elements (Benoit, 1995 and references therein). Benoit (1995) calculated the “true” partition coefficient for lead to be greater than $10^{7.4}$ (compared to partition coefficients of $\sim 10^5$ to 10^8 for filter retained/filtrate lead), indicating that truly dissolved lead concentrations were extremely low.

Lead in Sediments

In anaerobic lake sediments, relatively volatile organo- (tetramethyl) lead may form through biological alkylation of organic and inorganic lead compounds (U.S. EPA, 1979, cited in ASTDR, 1997).

III. Effects Characterization

This section, along with the bioaccumulation potential section, is subdivided to evaluate receptors of the freshwater and terrestrial ecosystems separately. Figure J-1 summarizes the range of effects data for receptors of concern illustrating the sensitivity of various taxa to exposure. For reference, the water quality standards for freshwater communities (NAWQC or secondary values) are included for both acute and chronic endpoints. These values can be disregarded for receptors in the terrestrial community, because the NAWQC only provides protection for aquatic receptors not predators of aquatic biota. NAWQC provide a context for effects ranges in the aquatic community.

Freshwater Ecosystem

Lead is toxic to aquatic biota, though effects are significantly modified by various factors. Waterfowl suffering from lead intoxication exhibit symptoms such as lethargy and emaciation (chemical form unknown). In birds, death usually is indirectly caused by starvation and vulnerability to predation (Eisler, 1988). Acute exposures of lead to aquatic invertebrates and fish of 1 to 500 mg/L have lethal effects; chronic exposures of 0.007 to 0.020 mg/L can have lethal effects (chemical form unknown) (Demayo et al., 1982). Aquatic invertebrate species in general show a wide range of sensitivity to lead exposures (Demayo et al., 1982). Some studies have found populations that have acclimated to high levels of lead (Demayo et al., 1982). Evidence suggests that populations growing in water with lead concentrations greater than 0.08 mg/L are sensitive to episodic acute exposures, as found in industrial discharges and mining discharges (Demayo et al, 1982). Chronic exposures of 0.019 mg/L have been found to increase mortality rate in the marsh snail (*Lymnaea palustris*) (Demayo et al., 1982). Adverse effects on daphnid reproduction have been observed at 0.001 mg Pb^{2+} /L (Eisler, 1988). In fish, lethal solutions of lead promote the formation of increased mucus, which coagulates over the entire body and gills, resulting in eventual suffocation (Eisler, 1988b). Developmental defects are reported in rainbow trout at levels of 7.6 $\mu\text{g/L}$ for a 19-month exposure period (Davies et al., 1976). Effects of lead poisoning in amphibians include the alteration of blood chemistry,

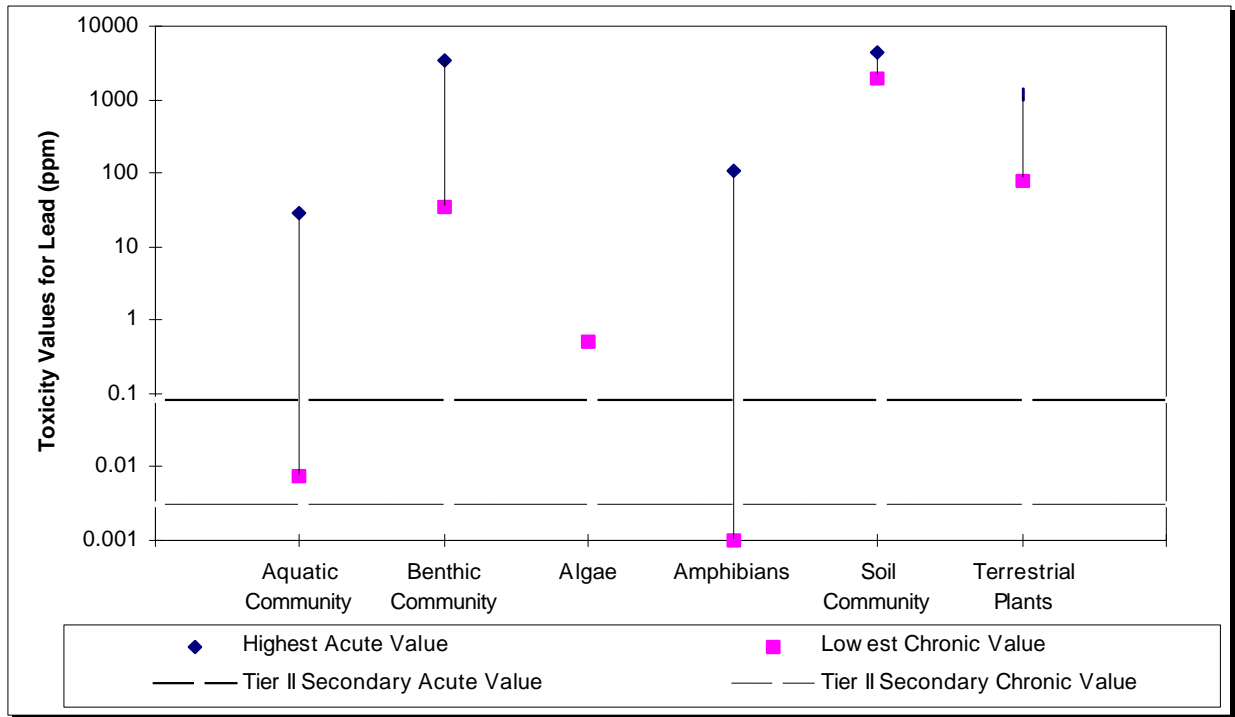


Figure J-1. Lead: effects ranges for selected ecological receptors.

sluggishness, vision impairment, and sloughing of skin (Eisler, 1988; Pain, 1995). Exposure of embryonic toads, *Xenopus laevis*, to static concentrations of 0.001 mg/L resulted in deformation of 82% of the population and 18% mortality, whereas 10 mg/L resulted in 100% mortality (Power et al., 1989).

Terrestrial Ecosystem

Lead acts at the molecular level to inhibit enzymes necessary for normal biological function in a variety of biota. In mammals, lead toxicity may affect the hematological system, the brain and nervous system, learning and behavior, and reproduction (Pain, 1995). In cattle, studies suggest that acute sublethal or lethal poisoning generally occurs at doses of 5 to 7 mg/kg-day (Pain, 1995). Decreases in survival rates in mice have been reported at drinking water exposures of 5 mg/L (Demayo et al., 1982). In rats, oral doses of 0.01 to 0.02 mg/kg-day have been associated with reproductive impairment and neurological problems (Hilderbrand et al., 1973 ; Krasovskii et al., 1979). Lead may also weaken an organism's immune system, even when no other signs of lead toxicity are observed (Pain, 1995). In birds, reproductive and developmental effects include decreases in egg production at 1.53 mg/kg-day oral exposures in Japanese quail and inhibited growth rates at 125 mg/kg-day in the American kestrel (Edens and Garlich, 1983; Pain, 1985).

Damage to plants with elevated lead contents is often negligible, but does vary among species (Eisler, 1988). Lead can have deleterious effects on plants at current lead levels in urban areas (Eisler, 1988). The decline of some European spruce forests has been attributed to

excessive concentrations of atmospheric lead (Pain, 1995). Reported effects include inhibition of plant growth and reductions in pollen germination, seed viability, and rates of photosynthesis and transpiration (Pain, 1995).

IV. Bioaccumulation Potential

Freshwater Ecosystems

Lead inhibits photosynthesis and ATP synthesis in plants and has been found to affect algal growth (Demayo et al., 1982). When grown in a diluted nutrient medium in the presence of 0.1 mg Pb/L, algal growth was reduced by 64%. Water quality parameters (e.g., pH) may all affect aquatic plants' sensitivity to lead (Demayo et al., 1982). Lead is bioconcentrated by aquatic organisms, but there is little evidence of biomagnification through the food chain (ATSDR, 1997). Lead concentrations tend to decrease with increasing aquatic trophic level, with the highest levels found in benthic organisms and algae and the lowest in upper trophic level predators (Eisler, 1988). A bioaccumulation factor (BAF) of 45.7 L/kg for fish was used to predict food chain exposures for piscivorous mammals and birds (unspecified chemical form) (Stephan, 1993). The value is based on a whole-body measured BAF of bluegill sunfish (*Lepomis macrochirus*).

Terrestrial Ecosystems

Sufficient data were not identified to determine bioconcentration factors (BCFs) for terrestrial vertebrates or terrestrial invertebrates. The uptake factors of soil to worm of 4.0E-2, and 3.0E-2 (kg soil/kg tissue) were used to arrive at a geometric mean of 3.5E-2 kg soil/kg tissue. These values are the ratios of lead in the earthworm, *Eisenia foetida*, to the concentration in sludge (Hartenstein et al., 1980). Davies (1983) reported values of 0.01 to 2.7 kg soil/kg tissue; however, the study from Davies (1983) was not used because the soil and sludge applied to the earthworms contained multiple chemical stressors. A soil-to-whole plant uptake value of 4.5E-2 was taken from an empirical study of lead uptake in natural forage; it is calculated by multiplying the plant uptake slope by a factor to convert the plant uptake slope to (μg constituent/g-plant tissue)/(kg-constituent/g-soil) (U.S. EPA, 1992).

V. Criteria Development

The benchmark values presented in this section for mammals and birds were used to derive protective media-specific criteria as outlined in the stressor-response profile methodology (i.e., analysis phase of ERA). By scaling the benchmark study by body weight to a representative wildlife receptor (e.g., rat study extrapolated to a shrew) and determining the dietary preferences of wildlife receptor and the potential bioconcentration in prey, a protective concentration (i.e., criteria) in soil, plants, or surface water was developed. Since criteria for receptors other than mammals and birds were already in media concentrations, this same derivation process was not required. A summary of criteria is provided in Table J-1. Although criteria were developed for numerous wildlife receptors of both the aquatic (e.g., otter, mink, and great blue heron) and terrestrial ecosystems (e.g. shrew, fox, and hawk), only the lowest criterion is presented in Table J-1. It is assumed that by protecting the more sensitive species, the other receptors are protected as well.

Table J-1. Lead Criteria in Soil, Sediment, Surface Water, and Plant Tissue Developed for Each Representative Receptor

Receptor	Criteria	Units	Exposure Pathway	Representative Species	Reference
Aquatic					
Mammals	3.0E-04	mg/L water	Food web	River Otter	Krasovskii et al., 1979
Birds	9.0E-04	mg/L water	Food web	Kingfisher	Edens and Garlich, 1983
Algae and Aquatic Plants	5.0E-01	mg/L water	Direct contact	<i>Chlorella vulgaris</i> and others	Suter and Tsao, 1996
Freshwater Community					
Total	3.2E-03	mg/L water	Direct contact	Aquatic biota	U.S. EPA, 1985
Dissolved	2.5E-03	mg/L water	Direct contact	Aquatic biota	U.S. EPA, 1985; 60FR22229
Benthic Community	3.0E+01	mg/kg sediment	Direct contact	Benthos	MacDonald, 1994
Amphibians (acute effects)	2.1E+00	mg/L water	Direct contact	Various amphibian species	Power et al., 1989; Schuytema and Nebekar, 1996
Terrestrial					
Mammals	4.7E-01*	mg/kg soil	Food web	Raccoon	Krasovskii et al., 1979
Birds	1.6E-01*	mg/kg soil	Food web	American woodcock	Edens and Garlich, 1983
Mammals	2.4E-02	mg/kg plant tissue	Food web	Meadow vole	Krasovskii et al., 1979
Birds	2.9E-01	mg/kg plant tissue	Food web	Northern bobwhite	Edens and Garlich, 1983
Plant Community	5.0E+01	mg/kg soil	Direct contact	Sycamore, red oak	Efroymsen et al., 1997
Soil Community	2.8E+01	mg/kg soil	Direct contact	Soil invertebrates	a

* This criterion should not be used because it is below soil background concentrations (lowest mean background concentration 16 mg lead/kg soil) . This may be an artifact of our backcalculation method (i.e., calculating media-specific criteria from the benchmark study).

^a Bengtsson et al., 1985; Bengtsson et al., 1986; Denneman and van Straalen, 1991; Marigomez et al., 1986; van de Meent et al., 1990.

Mammals: Numerous studies were identified that addressed the effects of lead in mammals. In an experiment lasting 20 to 30 days, rats were administered lead in oral doses of 0.05, 0.005, and 0.0015 mg/kg-d (Krasovskii et al., 1979). Impairment of the functional capacity of the male rat's spermatozoa was observed in rats receiving the maximum dose of 0.05 mg/kg-day. The gonadotoxic effects at 0.05 mg/kg-day resulted in an inferred NOAEL of 0.005 mg/kg-day. In another experiment in the same study, male and female rats were given the same doses of lead as above for 6 to 12 months. Neurological deficits, including disruption of conditional responses and motor activity, were observed at 0.05 and 0.005 mg/kg-day.

The NOAEL for gonadotoxic effects from the Krasovskii et al. (1979) study was chosen to derive the toxicological benchmark for the following reasons: (1) doses were administered over a chronic duration and via oral ingestion, an ecologically significant exposure pathway; (2) it focused on irregularities in the male rat's reproductive system as a critical endpoint; (3) it contained dose response information; and (4) it resulted in the lowest toxicity value for a critical endpoint.

In another investigation, dogs that were given a single dietary dose of 0.32 mg/kg-day for an unspecified period of time exhibited clinical signs of chronic lead toxicity (Demayo et al., 1982). Also, Hilderbrand et al. (1973) treated male and female rats with oral doses of lead of 5 and 100 µg/day for 30 days. Gonadotoxic effects in both the male and female rats were observed at the 100-µg/day dose resulting in an inferred NOAEL of 5 µg/day. To obtain the NOAEL as a daily dose, the reported dose was divided by the geometric mean (0.235 kg) of the male and female rats' reported body weights, resulting in a daily dose of 0.02 mg/kg-day.

The study by Hilderbrand et al. (1973) was not selected for the derivation of a benchmark because it did not report the lowest toxicity value for a critical endpoint. The Demayo et al. (1982) study was not chosen because of the absence of sufficient dose-response information and lack of critical endpoints.

The same surrogate-species study (Krasovskii et al., 1979) was used to derive the lead benchmark for mammalian species representing the terrestrial ecosystem.

Birds: There were several studies that investigated the effects of lead toxicity on birds. In a series of experiments, Edens and Garlich (1983) monitored the egg production of chickens and Japanese quail. Results showed that Japanese quail are more sensitive than chicken hens. When the lowest dose of 1 mg Pb/kg feed was administered for 5 weeks from day of hatch, egg production in Japanese quail was significantly reduced. This resulted in a reported LOAEL of 1 mg/kg-feed. This corresponds to a daily dose of 0.21 mg/kg-day based on a body weight value of 0.150 kg and a food intake value of 0.031 kg/day, both obtained from the study. In the absence of an experimental NOAEL, the NOAEL used is extrapolated from LOAEL of 0.21 mg/kg-day by a factor of 10 to arrive at an estimated NOAEL of 0.021 mg/kg-day.

The LOAEL reported by Edens and Garlich (1983) for Japanese quail was selected to derive the avian benchmark value for the freshwater ecosystem. This study was chosen for the following reasons: (1) doses were administered via oral ingestion, an ecologically significant exposure pathway; (2) it focused on reproductive toxicity as a critical endpoint; (3) it contained dose response information; and (4) it resulted in the lowest toxicity value for a critical endpoint.

Growth rate suppression occurred in chickens exposed to 1,850 ppm of dietary lead for 4 weeks (Franson and Custer, 1982). Conversion of this dose into units of mg/kg-day required the use of an allometric equation for chickens (U.S. EPA, 1988):

$$\text{Food consumption (kg/day)} = 0.075(W^{0.8449})$$

where W is body weight in kilograms. Based on the geometric mean of reported body weights of 0.110 kg for the control birds and the derived food consumption rate of 0.012 kg/day, the 1,850-ppm dose corresponds to a daily dose of 202 mg/kg-day. In another study, American kestrels exposed to doses of 10 and 50 ppm for 6 months exhibited no impairment of survival, egg laying, fertility, or eggshell thickness, suggesting a NOAEL of 50 ppm (Pattee, 1984). Conversion of this dose into units of mg/kg-day required the use of an allometric equation for birds (Nagy, 1987):

$$\text{Food consumption (g/day)} = 0.648(W^{0.651})$$

where W is body weight in grams. Using a reference kestrel body weight of 120 g (U.S. EPA, 1993) and a calculated food consumption rate of 15 g/day, the 50-ppm dose was converted to a daily dose of 6.3 mg/kg-day. In another study, Pain (1985) examined the growth of 1-day-old American kestrel nestlings exposed orally to 25, 125, and 625 mg/kg-day of dietary lead. The authors reported a NOAEL of 25 mg/kg-day and a LOAEL of 125 mg/kg-day.

The other studies mentioned above were not selected, either because they did not focus on a reproductive endpoint or because they lacked sufficient dose-response information.

Freshwater Community: Two sources were evaluated in selecting criteria for the protection of aquatic biota: (1) Final Chronic Values (FCV) derived under the Great Lakes Water Quality Initiative (GLWQI) (U.S. EPA, 1995) and (2) National Ambient Water Quality Criteria (NAWQC) published by the EPA Office of Water. The FCV of 3.2E-03 mg/L for lead and developed under the NAWQC was selected as the appropriate criteria to use in this analysis because no criteria were available for lead under GLWQI work (U.S. EPA, 1985). The GLWQI value was considered preferable to the NAWQC because: (1) the GLWQI value is based on the same methodology used to develop NAWQC (i.e., Stephan et al., 1985); (2) the NAWQC data set was augmented with previously unavailable acute and chronic toxicity data; and (3) species taxa used to generate the GLWQI values are suitable for national application since they include species and taxa found throughout the United States. But lacking the GLWQI value for lead, the NAWQC was used. It should be noted that the toxicity of lead is hardness dependent; therefore, the FCV (in µg/L) was calculated using the following equation (U.S. EPA, 1995), assuming a water hardness of 100 mg/L as calcium carbonate (CaCO₃):

$$e^{(1.273(\ln \text{ hardness}) - 4.705)}.$$

Although total concentrations of metals are still deemed scientifically defensible by EPA, recent EPA guidance recommends the use of dissolved metal concentrations to better reflect the bioavailability of metals (e.g., Prothro, 1993). Consequently, the FCV for lead was adjusted to provide dissolved concentrations as described in 60 FR 22231 (*Water Quality Standards...Revision of Metals Criteria*). The lead FCV was adjusted using a conversion factor (CF) of 0.791 for chronic effects to give a dissolved surface water criterion of 2.5E-03 mg/L.

This adjustment reflects the current EPA position on criteria development and regulatory application of metals; however, the issue of metal bioavailability in surface waters is the topic of intensive research (e.g., Bergman and Dorward-King, 1997). For example, the relationship between water characteristics (e.g., dissolved organic matter), copper bioavailability, and toxicity has been investigated in some detail (e.g., Allen and Hansen, 1996). For completeness, the total and dissolved surface water criteria are presented in Table 1 even though the values are identical.

Amphibians: No suitable subchronic or chronic studies were identified for criteria development that studied the effects of lead toxicity on reproductive or developmental endpoints in amphibian species. The variability between experimental designs and test endpoints made consistent comparisons between chronic data prohibitive; however, both acute and chronic data were identified to characterize the toxicity of lead to amphibian species. Review of data collected from six experiments indicate that the acute toxicity of lead ranges from 0.04 to 105 mg/L, with a geometric mean of 2.1 mg/L. Acute and chronic studies were conducted on various amphibian species (i.e., 11 amphibian species represented) during embryo, tadpole, and adult lifestages. Developmental deformities were noted in embryos of *Xenopus laevis* exposed to lead concentrations of 1 to 3 mg lead/L. Other behavioral responses to lead exposure are indicated at concentrations ranging from 0.5 to 1 mg lead/L. The observation that the lowest acute amphibian value approximates (i.e., within a factor of 2) the FAV of 0.082 mg lead/L determined for the freshwater community indicates that a large percentage of amphibian species may be protected at concentrations protective of the aquatic community. Investigations are ongoing to review the possibility of incorporating amphibian data into the NAWQC. Since amphibian species are more likely to breed in standing waters such as wetlands or ponds, the appropriateness of combining protective levels of amphibian receptors and the freshwater community is unclear at this time (Power et al., 1989; Schuytema and Nebekar, 1996).

Algae and Aquatic plants: Relevant endpoints for aquatic plants focused on the ability of plants to support higher trophic levels as well as the ability to provide habitat for other species in the freshwater ecosystem. The benchmarks for aquatic plants were either: (1) a no observed effects concentration (NOEC) or a lowest observed effects concentration (LOEC) for vascular aquatic plants (e.g., duckweed) or (2) an effective concentration (EC_{xx}) for a species of freshwater algae, frequently a species of green algae (e.g., *Selenastrum capricornutum*). For lead, the benchmark value was determined to be 5.0E-01 mg/L based on the growth inhibition of *Chlorella vulgaris*, *Scenedesmus quadricauda*, and *Selenastrum capricornutum* (Suter and Tsao, 1996). Moderate confidence is placed in this criterion since it is only based on several studies.

Benthic Community: The premier source of field sediment data is the National Oceanic and Atmospheric Administration (NOAA), which annually collects and analyzes sediment samples from sites located in coastal marine and estuarine environments throughout the United States as part of the National Status and Trends Program (NSTP). From the range of adverse effects data, criteria are developed estimating the 10th percentile effects concentration (ER-L) and a median effects concentration (ER-M) for adverse effects in the sediment community (Long et al., 1995). These values are not NOAA standards; rather, they are used to rank sites based on the potential for adverse ecological effects. A second criteria document evaluated for sediment criteria development was the *Approach to the Assessment of Sediment Quality in Florida Coastal Waters Volume 1- Development and Evaluation of Sediment Quality Assessment Guidelines* (MacDonald et al., 1994) published by the Florida Department of Environmental Protection

(FDEP). The criteria developed by FDEP were also based on the NOAA data; however, the method of derivation of the criteria was changed. FDEP calculated the criteria (i.e., threshold effects level, TEL) from the geometric mean of the 50th percentile of no effects data and the 15th percentile of the low effects data. The NOAA data, used in both documents, are based on total metal concentrations in sediments, and the toxicity endpoints were measured on species of amphipods, arthropods, and bivalves in addition to a variety of community-based endpoints (e.g., abundance, mortality, species composition, species richness). The FDEP criterion was chosen above the NOAA criterion for the following reasons; (1) the same database was used for both the NOAA criteria and the FDEP criteria development, only different derivation methods were used; (2) in most cases, the FDEP criterion was more conservative than the NOAA criteria because a larger portion of the low effects data was used in benchmark development; and (3) the marine TELs developed by the FDEP were found to be analogous to TELs observed in freshwater organisms (Smith et al., 1996).

The criterion for lead was derived from 402 toxicity data points for low and no effects levels. For the screening level analysis of lead, the TEL of 3.0E+01 mg lead/kg sediment was selected as an appropriate sediment criterion. Based on the quality and quantity of lead sediment data, the degree of confidence in the TEL value for lead was considered high (MacDonald, 1994).

Terrestrial Plants: As presented in Efroymson et al. (1997), phytotoxicity benchmarks were selected by rank-ordering the lowest observable effects concentration (LOEC) values and then approximating the 10th percentile. If fewer than 10 studies were available, the lowest LOEC was selected as the benchmark. Such LOECs applied to reductions in plant growth, yield, or seed elongation, or other effects reasonably assumed to impair the ability of a plant population to sustain itself. The selected benchmark for phytotoxic effects of lead in soils is 50 mg lead/kg soil (Efroymson et al., 1997). The derivation of the criterion is based on 17 phytotoxicity data points on various agricultural (e.g., barley, ryegrass) and silviculture (e.g., spruce) species measuring growth endpoints such as height and weight of shoots and roots, yield, and germination success. Considering this criterion was based on multiple studies over a range of species, confidence in this benchmark is high.

Soil Community: A community-based soil criteria was developed for lead using the methods presented in section 9.2.2.2 of the background document. The ecotoxicity data applied to the method are presented in Table J-6.

The value generated from this method resulted in a soil criterion of 28 mg lead/kg soil. This value, developed from no effect concentrations to various soil-based organisms, is more appropriate than criteria based on a single soil species such as earthworms. The criterion was derived to protect 95% of the species in the soil community providing protection to the long-term sustainability of a functioning soil community. Because five studies were used to derive this criteria, confidence in this criterion is moderate.

Table J-6. Data Set Used to Derive Soil Fauna Benchmark for Lead

Species	LOEC/ NOEC	Endpoint	Soil concentration (mg/kg)	Geometric mean (mg/kg)	Taxonomic grouping	Reference
Platynothrus peltifer	NOEC	reproduction	252		group 2	Denneman and van Straalen, 1991
Onychiurus armatus	NOEC	growth & reproduction	643		group 4	Bengtsson et al., 1985
Lumbricus rubellus	NOEC NOEC	reproduction growth	241 1133	523	group 5	van de Meent et al., 1990 van de Meent et al., 1990
Dendrobanea ribida	NOEC NOEC	reproduction reproduction	797 803	800	group 6	Bengtsson et al., 1986 Bengtsson et al., 1986
Porcellio scaber	NOEC	reproduction	23.4		group 7	van de meent et al., 1990
Arion ater	NOEC	litter breakdown	586		group 8	Marigomez et al., 1986

References

- Allen, H.E., and D.J. Hansen. 1996. The importance of trace metal speciation to water quality criteria. *Water Environ Res* 68(1):42-54.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1997. *Toxicological Profile for Lead* (Update). Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- Bengtsson, G., T. Gunnarsson, and S. Rundgren. 1985. Influence of metals on reproduction, mortality and population growth in *Onychiurus armatus* (Collembola). *Journal of Applied Ecology* 22:967-978.
- Bengtsson, G., T. Gunnarsson, and S. Rundgren. 1986. Effects of metal pollution on the earthworm *Dendrobaena rubida* (Sav.) in acidified soils. *Water, Air, and Soil Pollution* 28:361-383.
- Benoit, G. 1995. Evidence of the particle concentration effect for lead and other metals in fresh waters based on ultraclean technique analysis. *Geochimica et Cosmochimica Acta* 59(13):2677-2687.
- Bergman, H.L., and E.J. Dorward-King (eds). 1997. *Reassessment of Metals Criteria for Aquatic Life Protection: Priorities for Research and Implementation*. SETAC Press, Pensacola, FL.
- Davies, B.E. 1983. Chapter 14: Heavy metal contamination from base metal mining and smelting: implications for man and his environment. In: *Applied Environmental Geochemistry*, I. Thornton (ed.), Academic Press, London. pp. 425 to 462.
- Davies, P.H., J.P. Goettl, Jr., J.R. Sinley, and N.F. Smith. 1976. Acute and chronic toxicity of lead to rainbow trout *Salmo gairdneri*, in hard and soft water. *Water Research* 10:199-206.
- Demayo, A., M.C. Taylor, K.W. Taylor, and P.V. Hodson. 1982. Toxic effects of lead and lead compounds on human health, aquatic life, wildlife plants, and livestock. *CRC Critical Reviews in Environmental Controls* 12(4):257-305.
- Denneman, C.A.J., and N.M. van Straalen. 1991. The toxicity of lead and copper in reproduction tests using the oribatid mite *Platynothrus peltifer*. *Pedobiologia* 35:305-311.
- Edens, F.W., and J.D. Garlich. 1983. Lead-induced egg production decrease in Leghorn and Japanese quail hens. *Poultry Science* 62:1757-1763.

- Efroymson, R.A., M.E. Will, G.W. Suter, II, and A.C. Wooten. 1997. *Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision*. ES/ER/TM-85/R3. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Lockheed Martin Energy Systems, Inc., Oak Ridge, TN.
- Eisler, R. 1988. Lead hazards to fish, wildlife, and invertebrates: a synoptic review. In: *Contaminant Hazard Reviews, Report No. 14*. U.S. Fish and Wildlife Service, U.S. Department of the Interior, Laurel, MD.
- Franson, J.C., and T.W. Custer. 1982. Toxicity of dietary lead in young cockerels. *Vet.Hum.Toxicol.* 24(6):421-423.
- Hart, B.T., and T. Hines. 1995. Trace elements in rivers. In: *Trace Elements in Natural Waters*, B. Salbu and E. Steinnes (eds.), CRC Press, Boca Raton, FL. pp. 203 to 221.
- Hartenstein, R., E.F. Neuhauser, and J. Collier. 1980. Accumulation of heavy metals in the earthworm *Eisenia foetida*. *Journal of Environmental Quality* 9(1):23-26.
- Hilderbrand, D.C., R. Der, W.T. Griffin, and M.S. Fahim. 1973. Effect of lead acetate on reproduction. *Am.J.Obstet.Gynecol.* 115(8):1058-1065.
- Krasovskii, G.N., L.Y. Vasukovich, and O.G. Chariev. 1979. Experimental study of biological effects of lead and aluminum following oral administration. *Environmental Health Perspectives* 30:47-51.
- Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environmental Management* 19(1):81-97.
- MacDonald, D.D. 1994. *Approach to the Assessment of Sediment Quality in Florida Coastal Waters. Volumes I & II*. Prepared for Florida Department of Environmental Protection, Office of Water Policy. Prepared by MacDonald Environmental Sciences Ltd., Ladysmith, British Columbia.
- Marigomez, J.A., E. Angulo, and V. Saez. 1986. Feeding and growth responses to copper, zinc, mercury, and lead in the terrestrial gastropod *Arion ater* (Linne). *Journal of Molluscan Studies* 52:68-78.
- Nagy, K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds. *Ecological Monographs* 57(2):111-128.
- Pain, D.J. 1995. Lead in the environment. In: *Handbook of Ecotoxicology*, D.J. Hoffman, B.A. Rattner, G.A. Burton, Jr., and J. Cairns, Jr. (eds.), Lewis Publishers, Boca Raton, FL. pp. 356 to 391.

- Pattee, O.H. 1984. Eggshell thickness and reproduction in American kestrels exposed to chronic dietary lead. *Archives of Environmental Contamination and Toxicology* 13:29-34.
- Power, T., K.L. Clark, A. Harfenist, and D.B. Peakall. 1989. A review and evaluation of the amphibian toxicological literature. In: *Technical Report Series No. 61*. Canadian Wildlife Service.
- Prothro, M.G. 1993. Office of Water Policy and Technical Guidance on Interpretation and Implementation of Aquatic Life Metals Criteria. Memorandum. U.S. Environmental Protection Agency,
- Santschi, P.H. 1988. Factors controlling the biogeochemical cycles of trace elements in fresh and coastal marine waters as revealed by artificial radioisotopes. *Limnology and Oceanography* 33(4, part 2):848-866.
- Schuytema, G.S., and A.V. Nebeker. 1996. *Amphibian Toxicity Data for Water Quality Criteria Chemicals*. EPA/600/R-96/124. National Health and Environmental Effects Research Laboratory, Western Ecology Division, U.S. Environmental Protection Agency, Corvallis, OR.
- Smith, S.L., D.D. MacDonald, K.A. Keenleyside, C.G. Ingersoll, and L.J. Field. 1996. A preliminary evaluation of sediment quality assessment values for freshwater ecosystems. *J. Great Lakes Res.* 22(3):624-638.
- Stephan, C.E. 1993. *Derivation of Proposed Human Health and Wildlife Bioaccumulation Factors for the Great Lakes Initiative*. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Duluth, MN.
- Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs. 1985. *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses*. PB85-227049. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Duluth, MN.
- Suter, G.W., II, and C.L. Tsao. 1996. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision*. ES/ER/TM-96/R2. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Health Sciences Research Division, Oak Ridge National Laboratory Risk Assessment Program, Oak Ridge, TN.
- U.S. EPA (Environmental Protection Agency). 1985. *Ambient Water Quality Criteria for Lead - 1984*. EPA 440/5-84-027. Criteria and Standards Division, Office of Water, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1988. *Recommendations for and Documentation of Biological Values for Use in Risk Assessment*. EPA/600/6-87/008. Office of Health and Environmental Assessment, Cincinnati, OH.

- U.S. EPA (Environmental Protection Agency). 1992. *Technical Support Document for Land Application of Sewage Sludge. Volume I*. EPA 822/R-93-001a. Office of Water, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1993. *Wildlife Exposure Factors Handbook. Volumes I and II*. EPA/600/R-93/187. Office of Health and Environmental Assessment and Office of Research and Development, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1995. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Aquatic Life in Ambient Water*. EPA-820-B-95-004. Office of Water, Washington, DC.
- van de Meent, D., T. Aldenberg, J.H. Canton, C.A.M. van Gestel, and W. Slooff. 1990. *Desire for Levels: Background Study for the Policy Document "Setting Environmental Quality Standards for Water and Soil"*. RIVM report no. 670101 002. National Institute of Public Health and Environmental Protection.

Ecotoxicological Profile for Ecological Receptors

Mercury

This ecotoxicological profile on mercury contains five sections: (1) background (e.g., background concentrations), (2) geochemistry of the constituent in various ecological media, (3) effects characterization, (4) bioaccumulation potential, and (5) criteria development. The first four sections are intended to provide an overview of the environmental factors that influence the toxicological potential of mercury so that the limitations of the criteria may be better understood. The fifth section presents the rationale and development of criteria for the suite of ecological receptors used to represent aquatic and terrestrial ecosystems. The profile is intended to present the ecotoxicological criteria in a broader environmental context, so the ecological significance of the criteria may be properly interpreted.

I. Background

Mercury occurs naturally as a mineral and is distributed throughout the environment by natural and anthropogenic processes. Natural processes include weathering of mercury-containing rocks and volcanic eruptions. Anthropogenic releases are primarily to the atmosphere. Major anthropogenic sources of mercury include mining; industrial processes involving the use of mercury, including chloralkali manufacturing facilities; combustion of fossil fuels, primarily coal; production of cement; and medical and municipal waste incineration. Background concentrations in soils range from less than 0.01 to 4.6 mg/kg soil (Dragun and Chiasson, 1991). Typical concentrations in uncontaminated river waters range from 0.1 to 0.5 µg/L with groundwater sources demonstrating the high end of this range. Sediments that can act as a sink for mercury contain background concentrations of 0.02 to 0.06 mg Hg/kg, although polluted sediments may have 0.1 to 746 mg Hg/kg (Eisler, 1987).

Mercury exposure has been linked to adverse effects to a multitude of species including plants, fish, aquatic invertebrates, birds, and mammals. In both aquatic and terrestrial plants, decreased growth, lethality, reduced photosynthesis, leaf injury (e.g., necrosis), and inhibition of metabolic enzymes has been reported. Aquatic receptors, such as fish and invertebrates, have demonstrated death, reduced reproduction, impaired growth and development, altered behavior and metabolic function. Avian and mammalian species demonstrate sublethal effects such as organ damage, decreased growth and reproduction, and behavioral modifications.

Mercury in the aquatic system is known to undergo microbially mediated biotransformation to form methylmercury, which is a more bioavailable and toxic compound than inorganic mercury in aquatic systems. Mercury bioaccumulates and biomagnifies up the

food chain creating potentially high exposures to piscivorous mammals and birds. Methylation of mercury results in significant exposure especially for receptors of the aquatic community, including those avian species that consume large quantities of fish in their diet (U.S. EPA, 1997).

II. Geochemistry of Mercury in Various Ecological Media

General

Mercury occurs naturally as a mineral and is distributed throughout the environment by natural and anthropogenic processes. Mercury can exist in three oxidation states, Hg^0 (elemental), Hg^+ (mercurous), and Hg^{2+} (mercuric). The most reduced form is elemental mercury (Hg^0), which is a liquid at ambient temperatures but readily vaporizes. Mercurous and mercuric mercury can form numerous inorganic and organic chemical compounds; however, mercurous mercury is rarely stable under ordinary environmental conditions.

Mercury is unusual among metals in that it tends to form covalent rather than ionic bonds. Most of the mercury encountered in the water/soil/sediments/biota (all environmental media except the atmosphere) is in the form of inorganic mercuric salts and organomercuries. Organomercuries are defined by the presence of a covalent C-Hg bond. This is thought to differ from the common behavior of inorganic mercury compounds associating with organic material in the environment. The compounds most likely to be found under environmental conditions are: the mercuric salts HgCl_2 , $\text{Hg}(\text{OH})_2$, and HgS ; the methylmercury compounds CH_3HgCl and CH_3HgOH ; and, in small fractions, other organomercuries (i.e., dimethylmercury and phenylmercury) (ATSDR, 1997).

- # Mercury can exist in the environment in three oxidation states: Hg^0 , Hg^+ , and Hg^{2+} .
- # Elemental mercury (Hg^0) readily vaporizes.
- # Mercurous mercury (Hg^+) is rarely stable under ordinary environmental conditions.
- # The compounds most likely to be found under environmental conditions are: the mercuric salts [HgCl_2 , $\text{Hg}(\text{OH})_2$, and HgS] and the methylmercury compounds [CH_3HgCl and CH_3HgOH].
- # Methylmercury is the most common organic form of mercury. It is soluble, mobile, and quickly enters the food chain.

Mercury in Soils

Average mercury concentrations in virgin and cultivated surface soils range from 20 to 625 ng/g. The highest concentrations are generally found in soils from urban locations and in organic versus mineral soils. The mercury content of most soils varies as a function of depth, with the highest mercury concentrations generally found in the surface layers.

Mercury is readily sorbed to soil substrates. It is strongly sorbed to humic materials in soils characterized by pH values equal to or greater than 4. It is also sorbed to iron oxides and clay minerals. Inorganic mercury sorbed to particulate material is not readily desorbed, and, as a consequence, leaching is relatively insignificant. Adsorption-desorption reactions with organic matter and soil minerals control soil pore water concentrations to very low levels.

Although mercury is thought to be strongly sorbed to the soil substrate, adsorption may be decreased, and mercury remobilized, as a function of increasing pH and/or chloride ion content. Mercuric mercury (Hg^{2+}) may form various complexes with chloride and hydroxide ions in soils. It is generally accepted that chloride is the most significant inorganic ligand responsible for increasing the mobility of mercury in the environment. This is due in part to chloride's abundance and persistence and the low affinity of mercury chloride complexes for soil surfaces. It is possible that other ligands, particularly other halides, could also cause a significant increase in mercury mobility.

Because mercury concentration is positively correlated to dissolved organic carbon, mercury may also be bound to humic and fulvic acids in soil pore water.

- # Mercury is strongly sorbed to soil substrates at pH values equal to or greater than 4.
- # Adsorption-desorption reactions with organic matter and soil minerals control soil pore water concentrations to very low levels.
- # Chloride concentration may be as important as pH in determining mercury mobility.
- # Mercury may also be mobilized through the reduction of ionic mercury to the more volatile elemental mercury and through methylation to form volatile organic compounds such as dimethylmercury.

Mercury may also be remobilized through the microbial reduction of Hg^{2+} to the more volatile elemental mercury (Hg^0) as well as the bioconversion to volatile organic forms (dimethylmercury). Because these reactions are generally biologically mediated, temperature and pH are important considerations. For example, volatilization is generally greater in warmer weather when soil microbial activity is greatest. Volatilization is also greater in acidic soils (pH values equal to or less than 3) (ATSDR, 1997).

Mercury in Surface Water

Most chemical analyses yield total mercury concentration for a given sample. Total mercury in water is made up principally of elemental mercury, dissolved complexes of methylmercury and mercuric ion, and particulate forms of methylmercury and mercuric ion. Total mercury is a poor predictor of mercury speciation. For example, methylmercury as a percent of total mercury in water ranges from a few percent to more than 60 percent and is not solely a function of total mercury concentrations in water.

Water samples collected from lakes and rivers in the Ottawa, Ontario, region of Canada had total mercury concentrations ranging from 3.5 to 11.4 ng/L, with organic mercury concentrations ranging from 22% to 37%. Higher concentrations were measured in water samples collected from Crab Orchard Lake in Illinois and from surface waters of lakes and rivers in California. Specifically, mercury measurements ranged from 70 to 281 ng/L for the Illinois samples and from 0.5 to 104.3 ng/L for the California samples.

Reactions with particulates dominate the fate of mercury in aquatic environments. In surface waters having an average concentration of sulfide, mercury will form mercuric sulfide (HgS) at pH ranges of 4 to 9. This compound is relatively insoluble in aqueous solutions and will precipitate out. Under acidic conditions, the activity of the sulfide ion decreases and the formation of mercuric sulfide is inhibited. Under these conditions, the formation of

methylmercury is favored instead. The formation of mercuric sulfide and the adsorption of mercury to particles result in a significant fraction of mercury settling to the bottom sediments.

Mercury can exist in surface water as both the mercuric (Hg^{2+}) and mercurous (Hg^+) states. Because mercurous mercury is unstable, mercuric mercury is the predominant form of the two. Under environmental conditions, mercuric ion forms dissolved organic and inorganic complexes in the water column.

Mercuric ion can be transformed by biological and/or photochemical reduction to elemental mercury (Hg^0) or by biological methylation to methylmercury (CH_3Hg^+). Once formed, elemental mercury can volatilize to the atmosphere, whereas methylmercury can be accumulated in the underlying sediments or bioaccumulated in the food web. These reactions are reversible, and mercuric ion can also result from the oxidation of elemental mercury or the demethylation of methylmercury.

Reduction of Hg^{2+} to Hg^0 can occur under both aerobic and anaerobic conditions. It is enhanced by light and inhibited by competition from chloride ions. Surface waters may be saturated with volatile elemental mercury at times; however, production is seasonal and the highest levels generally occur during the warmer summer months. The exchange of elemental mercury with the atmosphere can lower the surface water mercury burdens.

Because of methylmercury's toxicity and tendency to bioaccumulate, it is a very important species of mercury. While some evidence for abiotic methylation exists, mercury methylation in the environment is mediated principally by sulfate-reducing bacteria that occur in freshwater and marine sediments. High rates of methylation have been observed in anoxic sediment and water and at the thermocline of the stratified lakes and estuaries.

As a biologically mediated reaction, methylmercury formation is sensitive to factors that affect biological activity as well as the physicochemical factors that govern the availability of inorganic mercury. The most important of these factors are dissolved oxygen concentration, temperature, lake basin characteristics (e.g., depth, water retention time), pH, sulfate and sulfide concentration, chloride concentration, water hardness, biological productivity, and total mercury concentration. Methylmercury production generally increases under conditions of elevated temperature and reduced dissolved oxygen concentration. In the anoxic hypolimnion of seasonally stratified lakes, methylmercury has been observed to accumulate at levels greater than

- # Mercury participates in a dynamic biogeochemical cycle in aquatic environments.
- # In aquatic environments having a pH range typical of environment conditions, the formation of mercuric sulfide (HgS) is favored. Mercuric sulfide precipitates out of solution, thus removing mercury from the water column.
- # Dissolved-phase mercuric complexes (HgCl_2) are important in the water column as they increase mobility.
- # Ionic mercury can be reduced to elemental mercury. Once formed, elemental mercury can volatilize, thereby reducing the dissolved phase mercury burden.
- # Ionic mercury can also be methylated to form methylmercury. This reaction is especially prevalent under anoxic conditions. Methylmercury tends to accumulate in the underlying sediments, also decreasing the dissolved phase mercury burden.

10 ng/L. This buildup has been related to in situ methylmercury production and remobilization from particulate matter (ATSDR, 1997).

Mercury in Sediments

Mercury levels in surface sediments of the St. Louis River range from 18 to 500 ng/L. Mercury was detected in sediment samples from Crab Orchard Lake in Illinois at greater than 60 $\mu\text{g/L}$. Surficial sediment samples from several sites along the Upper Connecting Channels of the Great Lakes had mercury concentrations ranging from below the detection limit to 55.80 $\mu\text{g/g}$. Mercury concentrations were correlated with particle size fractions and organic matter content.

- # Inorganic mercury tends to sorb to particulate matter and settle out. Inorganic mercury is not readily desorbed and the sediments are an important sink for both freshwater and estuarine systems.
- # Sediments are also considered to be a sink for methylmercury; however, methylmercury may be released back into the water column under anaerobic/sulfidic conditions.

The dominant process controlling the distribution of mercury compounds in the environment appears to be the sorption of nonvolatile forms to soil and sediment particulates, which settle out of the water column with little resuspension from the sediments back into the water column. Inorganic mercury sorbed to particulate material is not readily desorbed. Thus, sediments are an important repository for inorganic forms of mercury. Sediments tend to be a reservoir for mercury in both freshwater and estuarine systems.

Sediments generally are also considered to be a sink for methylmercury. In contrast to inorganic mercury, however, methylmercury may be released back into the water column under anaerobic/sulfidic conditions. Specifically, methylation is favored under anaerobic conditions, whereas demethylation is favored in oxic waters (ATSDR, 1997).

III. Effects Characterization

This section, along with the bioaccumulation potential section, are subdivided to evaluate receptors of the freshwater and terrestrial ecosystems separately. Figure J-2 summarizes the range of effects data for receptors of concern illustrating the sensitivity of various taxa to exposure. For reference, the water quality standards for freshwater communities (NAWQC or secondary values) are included for both acute and chronic endpoints. These values can be disregarded for receptors in the terrestrial community, because the NAWQC only provides protection for aquatic receptors, not predators of aquatic biota. The NAWQC provide a context for effects ranges in the aquatic community.

Freshwater Ecosystems

Aquatic organisms (e.g., fish and aquatic invertebrates) and predators (e.g., mammals and birds) that characteristically forage in freshwater ecosystems are sensitive to mercury exposures. Because biotransformation of inorganic mercury to methylmercury occurs primarily in sediments, higher concentrations of methylmercury are usually measured in surface water and

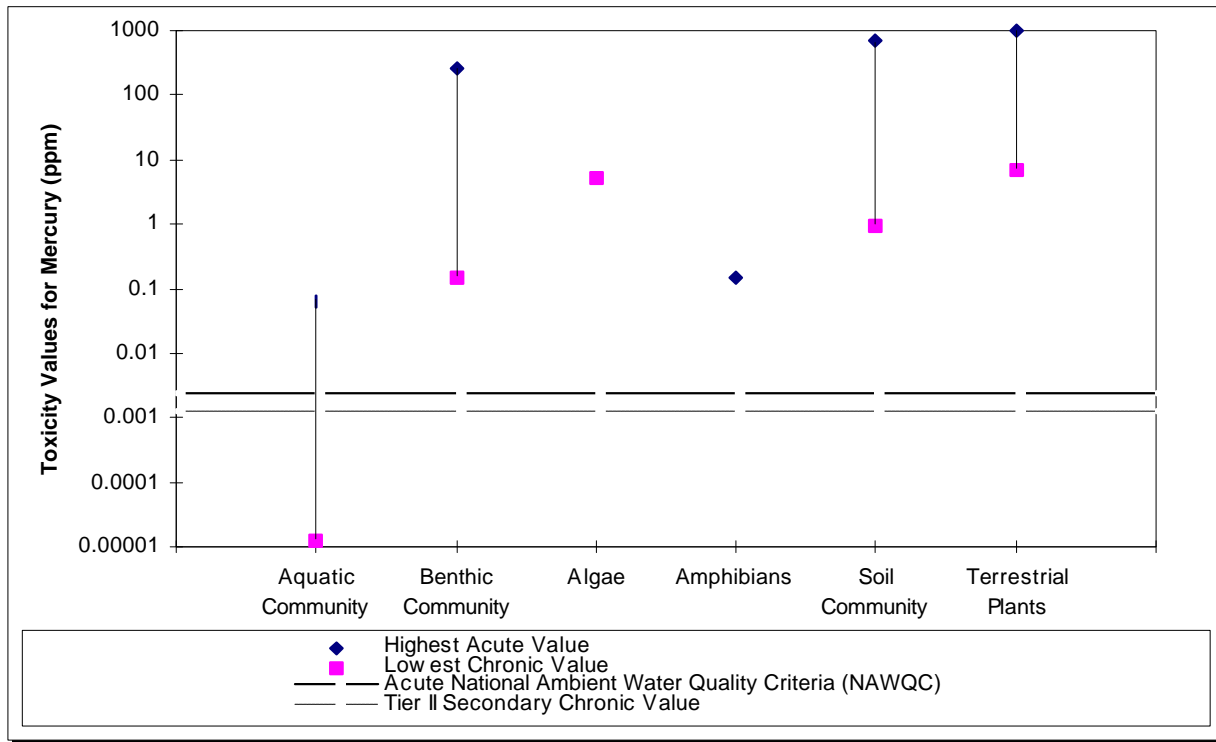


Figure J-2. Mercury: effects ranges for selected ecological receptors.

sediment media. Given that methylmercury is more toxic and bioaccumulative than inorganic mercury, the potential for adverse effects in freshwater ecosystems is high.

Acute toxicity in the aquatic community for inorganic mercury ranges from 5 to 10 $\mu\text{g/L}$ and 155 to 440 $\mu\text{g/L}$ for freshwater invertebrates and fish, respectively. In contrast, for organic methylmercury, acute toxicity has been documented to range from 5.0 to 65 $\mu\text{g/L}$ for yearling brook trout, and, for invertebrates, acute effects have ranged from 0.9 to 3.2 $\mu\text{g/L}$. In both inorganic mercury and methylmercury, acute effects in fish included behavioral changes and lethality. For chronic effects, concentration at 0.04 $\mu\text{g/L}$ and 0.79 $\mu\text{g/L}$ reduced the growth of rainbow trout and brook trout, respectively (Eisler, 1987). A relative comparison of the acute toxicity observed in fish and aquatic invertebrates from exposure to inorganic mercury and methylmercury reveals that methylmercury is more toxic by approximately an order of magnitude.

Among mercury species, methylmercury is the most toxic to mammals. Daily doses of methylmercury ranging from 0.1 to 0.5 mg/kg-day or 1.0 to 5.0 mg/kg diet were lethal to sensitive mammals (Eisler, 1987). Central nervous system toxicity, weight loss, and mortality were observed among rats fed a diet containing 250 mg/kg methylmercury for 2 weeks (Verschuuren et al., 1976a). Rats consuming 2.5 mg/kg methylmercury in the diet for 2 years displayed adverse impacts to growth and physiological functions (Verschuuren et al., 1976b). No adverse effects to reproductive endpoints were observed in rats fed at 0.5 mg/kg and below over a three-generation experiment, but, at 2.5 mg/kg, offspring survival rate was reduced.

For birds, acute toxicity for methylmercury ranges from 2.2 to 23.5 mg/kg for mallard (*Anas platyrhynchos*), 11.0 to 27.0 mg/kg diet for Japanese quail (*Coturnix japonica*), and 37.9 mg/kg for whistling duck (*Dendrocygna bicolor*). Heinz (1979) fed mallard ducks a diet containing 0.5 mg/kg methylmercury for three generations. Although it did not affect adult weights or weight changes, for those female birds exposed to methylmercury, decrease in clutch number, egg shell thickness, and behavioral modifications in young were noted.

Algae are more resistant to mercury exposure than other aquatic receptors. Toxic effects were evident in algae and submerged aquatic vegetation exposed to inorganic mercury (2+) at concentrations ranging from 53 to 3,400 µg/L. In algae exposed, methylmercury toxicity was reported to range from 0.8 to 6.0 µg/L (U.S. EPA. 1997). In contrast, amphibians demonstrate higher sensitivity. Exposure of developing toad eggs to 3 mg/L mercuric chloride resulted in 100% mortality. Further, developmental effects in *X. laevis* eggs were noted at 0.146 ppm (Power et al., 1989). No chronic studies of extended duration could be identified for amphibian populations. However, given the observed levels of acute toxicity in amphibian species, amphibians are likely to demonstrate sensitivity within the range of fish populations (Figure J-2).

Terrestrial Ecosystems

In evaluating the effects of mammals and birds characteristic of terrestrial ecosystems, the potential ecotoxicological effects resulting from exposure to contaminated prey were assumed to be similar to those discussed for mammals and birds representative of freshwater ecosystems. Although uptake parameters are modified to reflect the different routes of exposure in terrestrial ecosystems, the toxicological response to mercury exposure will be similar across these taxa.

Terrestrial plants and soil invertebrates appear to be less sensitive to mercury exposure than other terrestrial receptors, which may be the direct result of how chemical equilibria in soils will influence mercury speciation (i.e., methylmercury is present in soils but not to the extent that it occurs in sediments). However, ecotoxicity data that fully characterize the effects through soil exposure routes to terrestrial plants and soil biota are limited. A number of studies were available that exposed terrestrial plants via solution; however, this exposure route did not adequately simulate probable environmental exposures; therefore, these data were not used.

IV. Bioaccumulation Potential

Freshwater Ecosystems

The bioaccumulative capacity of mercury in freshwater organisms, as methylmercury and inorganic mercury, is key to evaluating food web exposures. Studies have indicated that mercury bioaccumulates in freshwater ecosystems. In phytoplankton, methylmercury bioconcentration factors (BCFs) of 90,000 to 107,000 (L/kg) have been estimated; whereas, total mercury BCFs have been reported in the range of 2,000 to 40,000. The large variability in total mercury BCFs is likely associated with the percent methylmercury (the more bioavailable chemical species) present in the surface water. Moving through the food web, zooplankton accumulate mercury to a greater degree than phytoplankton. The literature indicates that BAFs range from 11,000 to 12,600,000 when zooplankton are exposed to methylmercury; whereas, zooplankton exposed to total mercury indicate BAFs ranging from 3,100 to 285,000 (U.S. EPA, 1997).

The BAFs reported in the *Mercury Study Report to Congress* (MRTC) (U.S. EPA, 1997) represent the current accepted approach to characterizing bioaccumulation in fish species. The BAFs reported in this document were used in the HWC-SERA analysis to estimate the bioaccumulation potential in trophic level 3 and trophic level 4 fish and subsequent exposures through the food web to piscivorous mammals and birds. The BAF values of 6,800,000 for trophic level 4 fish and 1,600,000 for trophic level 3 fish were used in estimating the mammalian and avian food chain exposures. The MRTC BAF values for fish were derived from field studies, which were preferred over laboratory exposures or modeled estimates. For an expanded explanation of the methodology and rationale for selection of the BAFs, refer to U.S. EPA (1997).

Terrestrial Ecosystem

Mercury bioaccumulation appears to be largely a problem associated with the aquatic ecosystem. Accumulation of total mercury in earthworms has been reported in BCFs ranging from 21.3 to 27.1 (Schuytema and Nebekar, 1996). Mercury bioaccumulation factors have been documented to be 11.1 in mink liver and 7.4 in kidney tissues (WHO, 1989). Average accumulation factors in terrestrial earthworms and small mammals have been reported at 5.2 and 0.12, respectively (Sample et al., 1998a, 1998b). Insufficient data were available to characterize uptake in terrestrial plants and other soil invertebrates.

V. Criteria Development

The benchmark values presented in this section for mammals and birds were used to derive protective media-specific criteria as outlined in the stressor-response profile methodology (i.e., analysis phase of ERA). By scaling the benchmark study by body weight to a representative wildlife receptor (e.g., rat study extrapolated to a shrew), determining the dietary preferences of wildlife receptor and the potential bioconcentration in prey, a protective concentration (i.e., criteria) in soil, plants, or surface water was developed. Since criteria for receptors other than mammals and birds were already in media concentrations, this same derivation process was not required. A summary of criteria is provided in Table J-2. Although criteria were developed for numerous wildlife receptors of both the aquatic (e.g., otter, mink, and great blue heron) and terrestrial ecosystems (e.g. shrew, fox, and hawk), only the lowest criterion is presented in Table J-2. By protecting the more sensitive species, other receptors are likely to be protected as well.

Mammals: Two subchronic studies were identified that reported dose-response data for mammalian wildlife. Rhesus monkeys were exposed to methylmercury chloride by gavage at doses of 0.05, 0.16, or 0.5 mg/kg-day during gestation days 20 through 30. No signs of malformation were seen at the two lower doses (Dougherty et al., 1974). However, the highest dose level was maternally toxic and abortient, suggesting a NOAEL of 0.16 mg/kg-day and a LOAEL of 0.5 mg/kg-day for reproductive effects.

A second study fed adult female mink rations containing methylmercury chloride at doses of 0.18, 0.29, 0.77, 1.3, and 2.4 mg/kg-day (Wobeser et al., 1976a,b). Groups exposed to doses of 0.29 to 2.4 mg/kg-day exhibited clinical signs of toxicity. The 0.18-mg/kg-day exposure group did not show clinical evidence of toxicity but did exhibit pathological alterations of the

**Table J-2. Mercury and Methylmercury Criteria in Soil, Sediment, and Surface Water
Developed for Representative Receptors**

Receptor	Criteria	Units	Exposure Pathway	Representative Species	Reference
Aquatic					
Mammals (total dissolved)	5.4E-07	mg/L water	Food web	River Otter	Wobeser et al., 1976a,b; U.S. EPA, 1997 Heinz, 1974; 1975; 1979; U.S. EPA, 1997
Birds (total dissolved)	4.2E-07	mg/L water	Food web	Kingfisher	
Mammals (methylmercury)	4.2E-08	mg/L water	Food web	River Otter	Wobeser et al., 1976a,b; U.S. EPA, 1997 Heinz, 1974; 1975; 1979; U.S. EPA, 1997
Birds (methylmercury)	3.3E-08	mg/L water	Food web	Kingfisher	
Algae and Aquatic Plants	5.0E+00	mg/L water	Direct contact	<i>Microcystis aeruginosa</i>	U.S. EPA, 1995a Suter and Tsao, 1996 MacDonald, 1994
Freshwater Community					
Mercury (II)	9.1E-04	mg/L water	Direct contact	Aquatic biota	
Methylmercury	2.8E-06	mg/L water	Direct contact	Aquatic biota	
Benthic Community	1.3E-01	mg/kg sediment	Direct contact	Benthos	
Terrestrial					
Mammals	3.8E+01	mg/kg soil	Food web	Raccoon	Wobeser et al., 1976a,b Heinz, 1974; 1975; 1979
Birds	1.5E-01	mg/kg soil	Food web	American woodcock	
Soil Community	1.0E-01	mg/kg soil	Direct contact	Soil invertebrates	Efroymson et al., 1997a

Insufficient data to develop a criterion for terrestrial plants

nervous system. The authors stated that clinical signs of toxicity in the 0.18-mg/kg-day exposure group would have probably emerged if the experiment had lasted longer. A LOAEL of 0.18-mg/kg-day was inferred for pathological alterations from this study. The NOAEL derived from this study was 0.055 mg/kg-day (U.S. EPA, 1997).

The NOAEL from the Wobeser et al. (1976a,b) study was selected to derive the toxicological benchmark because: (1) doses were administered over a chronic duration and via oral ingestion, an ecologically significant exposure pathway; (2) the study focused on toxicity endpoints that could impact the reproductive potential of a species; and (3) it contained adequate dose-response information. The Dougherty et al. (1974) study was also an adequate study for selection; however, the premier source of information on mercury's risk to ecological receptors (*Mercury Study Report to Congress*, U.S. EPA, 1997) considered the Wobeser et al. (1976a,b) to be a more appropriate benchmark study for criteria derivation.

Birds: Several studies were identified that investigated the effects of methylmercury on avian species. In a series of studies carried over three generations, Heinz (1974, 1975, 1976a, 1976b, 1979) assessed the effects of dietary methylmercury on mallard ducks. Adult mallard ducks given doses of 0.078 and 0.468 mg/kg-day (assuming an uptake rate of 156 g/kg bw/d) for up to 2 years were monitored for egg production, hatching success, and hatchling viability. Based on an assessment of percent cracked eggs, egg production, or number of eggs producing normal hatchlings, no significant reproductive effects were observed in the first generation. However, the survival rate of offspring from the 0.468 mg/kg-day treatment group was significantly lower. Second generation parents on the 0.078-mg/kg-day diet exhibited abnormal egg-laying behavior and impaired reproduction, and their ducklings had a slowed growth rate. Third generation hens in the 0.078-mg/kg-day treatment group laid fewer viable eggs than those in the control group. Behavior tests designed to measure approach response to maternal calls and avoidance response to a frightening stimulus pooled over three generations indicate the cumulative effects over three generations were significant at the lowest dose level. Therefore, a LOAEL of 0.078 mg/kg-day was inferred based on adverse reproductive and behavioral effects across the three generations of mallard ducks.

Ring-necked pheasants were exposed to dietary methylmercury at doses equivalent to 0.18, 0.37, and 0.69 mg/kg-day for 12 weeks (Fimreite, 1970). Reduced hatchability and egg production as well as increased numbers of shell-less eggs were reported at all dose levels. Based on these results, a LOAEL of 0.18 mg/kg-day can be inferred for reproductive effects. In another study by Fimreite (as cited in U.S. EPA, 1993), leghorn cockerel chicks were exposed to dietary methylmercury at concentrations of 1.1, 2.1, and 3.2 mg/kg-day for 21 days. A significant increase in mortality occurred at exposure to 3.2 mg/kg-day, while chicks maintained at 2.1 mg/kg-day exhibited decreases in growth. Although this study reports a NOAEL of 2.1 mg/kg-day for mortality and a LOAEL of 1.1 for growth, it is unclear as to whether these exposure levels would affect an entire population's survival. Reproductive effects were seen in white leghorn laying hens when they were exposed to methylmercury at dietary concentrations of 4.9 and 9.8 mg/kg-day for an unspecified period of time (Scott, 1977). Both dose levels severely impacted egg production and weight, fertility of eggs, hatchability of fertile eggs, and eggshell strength.

Although the studies by Fimreite (1970) and Scott (1977) provide reproductive endpoints in response to multiple, dietary methylmercury dose levels, the results of the Heinz (1974, 1975, 1976a, 1976b, 1979) multigeneration studies were found to be most appropriate for the estimation of a benchmark value for avian species. These studies provide reproductive and behavioral effects due to methylmercury exposure over three generations of mallards. From all the avian studies identified, Heinz (1974, 1975, 1976a, 1976b, 1979), furnish the most conservative dose level that could impair the survival and reproductive potential of an avian population. Therefore, the LOAEL of 0.078 mg/kg-day was used to derive a benchmark value for representative avian species of the freshwater ecosystem. As indicated in the *Mercury Study Report to Congress* (U.S. EPA, 1997), an uncertainty factor of 3 was applied to estimate a NOAEL of 0.026 mg/kg-d.

Data were available on reproductive, developmental, growth, and survival endpoints for methylmercury exposure. In addition, the data set contained studies that were conducted over acute and chronic durations and during sensitive life stages. Other than the studies discussed for the freshwater ecosystem, no avian toxicity data were identified. Therefore, the NOAEL of 0.026 mg/kg-day extrapolated from Heinz (1974, 1975, 1976a, 1976b, 1979) was chosen to calculate a benchmark value for the representative avian species in the terrestrial ecosystem.

Freshwater Community: Two sources were evaluated in selecting criteria for the protection of aquatic biota: (1) Final Chronic Values (FCV) derived under the Great Lakes Water Quality Initiative (GLWQI) (U.S. EPA, 1995a) and (2) National Ambient Water Quality Criteria (NAWQC) published by the EPA Office of Water. The FCV of 9.1E-04 mg/L for mercury (II) developed under the GLWQI was used. The GLWQI values were considered preferable to the NAWQC because: (1) the GLWQI values are based on the same methodology used to develop NAWQC (i.e., Stephan et al., 1985); (2) the NAWQC data set was augmented with previously unavailable acute and chronic toxicity data; and (3) species taxa used to generate the GLWQI values are suitable for national application since they include species and taxa found throughout the United States.

Sufficient data were not available to develop an FCV for methylmercury, rather a Secondary Chronic Value (SCV) of 2.8E-06 mg/L for methylmercury developed by Oak Ridge National Laboratory (Suter and Tsao, 1996) was selected as the appropriate criteria to use in this analysis. SCVs are calculated by methods analogous to those used to derive FCVs for both the GLWQI and NAWQC. However, when the eight data requirements for developing the FCV were not available, the SCV criterion was based on one to seven of the eight required criteria. The SCV for methylmercury was derived from four data points based on toxicity endpoints found in rainbow trout (*Oncorhynchus mykiss*) and brook trout (*Salvelinus fontinalis*). From these data, an SAV of 9.917E-5 mg/L and SACR of 35.72 were calculated. The resulting ratio of these values (i.e., SAV/SACR) determined the SCV of 2.8E-6 mg/L (Suter and Tsao, 1996).

Although total concentrations of metals are still deemed scientifically defensible by EPA, recent EPA guidance recommends the use of dissolved metals concentrations to better reflect the bioavailability of metals (e.g., Prothro, 1993). Consequently, the FCVs can be adjusted to provide dissolved concentrations as described in 60 FR22229-22237 (*Water Quality Standards: Establishment of Numeric Criteria for Priority Toxic Pollutants; States' Compliance—Revision of Metals Criteria*, U.S. EPA, 1995b); however, a CF was not available for mercury or

methylmercury. This adjustment (i.e., use of conversion factors) reflects the current EPA position on criteria development and regulatory application of metals; however, the issue of metal bioavailability in surface waters is the topic of intensive research (e.g., Bergman and Dorward-King, 1997). For example, the relationship between water characteristics (e.g., dissolved organic matter), copper bioavailability, and toxicity has been investigated in some detail (e.g., Allen and Hansen, 1996). Aquatic criteria developed in this section are summarized in Table J-2.

Amphibians: No suitable subchronic or chronic studies were identified that studied the effects of mercury toxicity on reproductive or developmental endpoints in amphibian species; however, several acute studies were identified characterizing mercury toxicity. Review of data collected from 67 experiments indicate that the acute toxicity of arsenic ranges from 0.001 to 108 mg mercury/L, with a geometric mean of 0.20 mg/L. Acute studies were conducted on various amphibian species (i.e., 27 amphibian species represented) during embryo, tadpole, and adult lifestages. Chemical exposures were conducted primarily with mercuric chloride. The observation that the lowest acute amphibian value approximates the FAV of 0.0024 mg mercury/L determined for the freshwater community indicates that some amphibian species may be sufficiently protected by the current acute freshwater criteria. A few chronic exposures were identified indicating deformity from 96-hour exposures to 0.0001 to 0.1 depending on the species. Longer exposures of 7 to 10 days indicate deformities at concentrations of 0.0003 to 0.08 mg mercury/L at varying degrees of severity and magnitude. Further, spermatogenesis was inhibited at concentrations of 0.3 mg mercury/L. Investigations are ongoing to review the possibility of incorporating amphibian data into the NAWQC. Since amphibian species are more likely to breed in standing waters such as wetlands or ponds, the appropriateness of combining protective levels of amphibian receptors and the freshwater community is unclear at this time (Power et al., 1989; Schuytema and Nebekar, 1996).

Algae and Aquatic plants: The toxicological benchmarks for aquatic plants were either: (1) a no observed effects concentration (NOEC) or a lowest observed effects concentration (LOEC) for vascular aquatic plants (e.g., duckweed) or (2) an effective concentration (EC_{xx}) for species of freshwater algae, frequently a species of green algae (e.g., *Selenastrum capricornutum*). For mercury the benchmark value was determined to be 5.0 mg/L based on the growth inhibition of *Microcystis aeruginosa*. Low confidence is placed in this criterion since it is only based on one study (Suter and Tsao, 1996).

Benthic Community: The premier source of field sediment data is NOAA, which annually collects and analyzes sediment samples from sites located in coastal marine and estuarine environments throughout the United States as part of the National Status and Trends Program (NSTP). From the range of adverse effects data, criteria are developed estimating the 10th percentile effects concentration (ER-L) and a median effects concentration (ER-M) for adverse effects in the sediment community (Long et al., 1995). These values are not NOAA standards; rather, they are used to rank sites based on the potential for adverse ecological effects. A second criteria document evaluated for sediment criteria development was the *Approach to the Assessment of Sediment Quality in Florida Coastal Waters Volume 1- Development and Evaluation of Sediment Quality Assessment Guidelines* (MacDonald et al., 1994) published by the Florida Department of Environmental Protection (FDEP). The criteria developed by FDEP were also based on the NOAA data; however, the method of derivation of the criteria was changed. FDEP calculated the criteria (i.e., threshold effects level, TEL) from the geometric

mean of the 50th percentile of no effects data and the 15th percentile of the low effects data. The NOAA data, used in both documents, are based on total metal concentrations in sediments, and the toxicity endpoints were measured on species of amphipods, arthropods, and bivalves in addition to a variety of community-based endpoints (e.g., abundance, mortality, species composition, and species richness). The FDEP criterion was chosen above the NOAA criterion for the following reasons; (1) the same database was used for both the NOAA criterion and the FDEP criteria development only different derivation methods were used; (2) in most cases, the FDEP criterion was more conservative than the NOAA criterion because a larger portion of the low effects data was used in benchmark development; and (3) the marine TEL developed by the FDEP were found to be analogous to TELs observed in freshwater organisms (Smith et al., 1996).

The criterion for mercury was derived from 331 toxicity data points for low and no effects levels. For the screening level analysis of mercury, the TEL of 1.3E-01 mg mercury/kg sediment was selected as an appropriate sediment criterion. Based on the quality and quantity of mercury sediment data, the degree of confidence in the TEL value for mercury was considered high (MacDonald, 1994).

Terrestrial Plants: As presented in Efroymson et al. (1997b), phytotoxicity benchmarks were selected by rank-ordering the LOEC values and then approximating the 10th percentile. If fewer than 10 studies were available, the lowest LOEC was selected as the criteria. Such LOECs applied to reductions in plant growth, yield, or seed elongation, or other effects reasonably assumed to impair the ability of a plant population to sustain itself. The proposed benchmark for phytotoxic effects of mercury in soils is 0.3 mg mercury/kg soil (Efroymson et al., 1997b). Since the criterion was based on a single study reporting unspecified effects and did not indicate the form of mercury applied to test soils or the terrestrial plant species exposed, this benchmark study was not appropriate for criteria development. No further studies were identified, so no criteria could be developed for the terrestrial plant community.

Soil Community: A soil benchmark was derived from the criterion proposed by ORNL (Efroymson et al., 1997a). The proposed criterion of 1.0E-01 mg total mercury/kg soil was the lowest toxicity value based on earthworm endpoints. Additionally, a microbial toxicity value was identified: 30 mg total mercury/kg soil. Value based on earthworm was proposed as the criterion because earthworm is an important component in promoting soil fertility, improving aeration and drainage of soil, and serving as an important food source for many higher trophic animals. Community-based criteria values should be used as they become available. Low confidence is placed in this criteria because of the lack of supporting data.

References

- Allen, H.E., and D.J. Hansen. 1996. The importance of trace metal speciation to water quality criteria. *Water Environ Res* 68(1):42-54.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1997. *Toxicological Profile for Mercury* (Draft). Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.

- Bergman, H.L., and E.J. Dorward-King (eds). 1997. *Reassessment of Metals Criteria for Aquatic Life Protection: Priorities for Research and Implementation*. SETAC Press, Pensacola, FL.
- Dougherty, W.J., F. Coulston, and L. Golberg. 1974. Toxicity of methylmercury in pregnant rhesus monkeys. *Toxicology and Applied Pharmacology* 39: 138. (Abstr.).
- Dragun, J., and A. Chiasson. 1991. *Elements in North American Soils*. Hazardous Materials Control Resources Institute, Greenbelt, MD. pp. 117 to 123.
- Efroymson, R.A., M.E. Will, and G.W. Suter, II. 1997a. *Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates and Heterotrophic Processes: 1997 Revision*. ES/ER/TM-126/R2. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Lockheed Martin Energy Systems, Inc., Oak Ridge, TN.
- Efroymson, R.A., M.E. Will, G.W. Suter, II, and A.C. Wooten. 1997b. *Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision*. ES/ER/TM-85/R3. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Lockheed Martin Energy Systems, Inc., Oak Ridge, TN.
- Eisler, R. 1987. Mercury hazards to fish, wildlife, and invertebrates: a synoptic review. In: *Contaminant Hazard Reviews, Report No. 10*. U.S. Fish and Wildlife Service, U.S. Department of the Interior, Laurel, MD.
- Fimreite, N. 1970. Effects of methyl mercury treated feed on the mortality and growth of leghorn cockerels. *Canadian Journal of Animal Science* 50:387-389.
- Heinz, G.H. 1974. Effects of low dietary levels of methyl mercury on mallard reproduction. *Bulletin of Environmental Contamination and Toxicology* 11(4):386-392.
- Heinz, G.H. 1975. Effects of methylmercury on approach and avoidance behavior of mallard ducklings. *Bulletin of Environmental Contamination and Toxicology* 13(5):554-564.
- Heinz, G.H. 1976a. Methylmercury: second-generation reproductive and behavioral effects on mallard ducks. *Journal of Wildlife Management* 40(4):710-715.
- Heinz, G.H. 1976b. Methylmercury: second-year feeding effects on mallard reproduction and duckling behavior. *Journal of Wildlife Management* 40(1):82-90.
- Heinz, G.H. 1979. Methylmercury: reproductive and behavioral effects on three generations of mallard ducks. *Journal of Wildlife Management* 43(2):394-401.
- Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environmental Management* 19(1):81-97.

- MacDonald, D.D. 1994. *Approach to the Assessment of Sediment Quality in Florida Coastal Waters. Volumes I & II*. Prepared for Florida Department of Environmental Protection, Office of Water Policy. Prepared by MacDonald Environmental Sciences Ltd., Ladysmith, British Columbia.
- Power, T., K.L. Clark, A. Harfenist, and D.B. Peakall. 1989. A review and evaluation of the amphibian toxicological literature. In: *Technical Report Series No. 61*. Canadian Wildlife Service.
- Prothro, M.G. 1993. Office of Water Policy and Technical Guidance on Interpretation and Implementation of Aquatic Life Metals Criteria. Memorandum. U.S. Environmental Protection Agency,
- Sample, B.E., J.J. Beauchamp, R.A. Efroymsen, and G.W. Suter, II. 1998a. *Development and Validation of Bioaccumulation Models for Small Mammals*. ES/ER/TM-219. Prepared for Office of Environmental Management, U.S. Department of Energy. Oak Ridge National Laboratory, Oak Ridge, TN.
- Sample, B.E., J.J. Beauchamp, R.A. Efroymsen, G.W. Suter, II, and T.L. Ashwood. 1998b. *Development and Validation of Bioaccumulation Models for Earthworms*. ES/ER/TM-220. Prepared for Office of Environmental Management, U.S. Department of Energy. Oak Ridge National Laboratory, Oak Ridge, TN.
- Schuytema, G.S., and A.V. Nebeker. 1996. *Amphibian Toxicity Data for Water Quality Criteria Chemicals*. EPA/600/R-96/124. National Health and Environmental Effects Research Laboratory, Western Ecology Division, U.S. Environmental Protection Agency, Corvallis, OR.
- Scott, M.L. 1977. Effects of PCBs, DDT, and mercury compounds in chickens and Japanese quail. *Federation Proceedings* 36(6):1888-1893.
- Smith, S.L., D.D. MacDonald, K.A. Keenleyside, C.G. Ingersoll, and L.J. Field. 1996. A preliminary evaluation of sediment quality assessment values for freshwater ecosystems. *J. Great Lakes Res.* 22(3):624-638.
- Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs. 1985. *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses*. PB85-227049. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Duluth, MN.
- Suter, G.W., II, and C.L. Tsao. 1996. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision*. ES/ER/TM-96/R2. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Health Sciences Research Division, Oak Ridge National Laboratory Risk Assessment Program, Oak Ridge, TN.

- U.S. EPA (Environmental Protection Agency). 1993. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife* (Proposed). EPA-822-R-93-007. Office of Water and Office of Science and Technology, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1995a. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,7,8-TCDD, and PCBs*. EPA-820-B-95-008. Office of Water, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1995b. Water quality standards: establishment of numeric criteria for priority toxic pollutants; states' compliance - revision of metals criteria. *Federal Register* 60 FR 22229-22237.
- U.S. EPA (Environmental Protection Agency). 1997. *Mercury Study Report to Congress. Volume VI - An Ecological Assessment for Anthropogenic Mercury Emissions in the United States*. EPA 452/R-97/008. Office of Air Quality Planning and Standards and Office of Research and Development, Washington, DC.
- Verschuuren, H.G., R. Kroes, E.M. Den Tonkelaar, J.M. Berkvens, P.W. Helleman, A.G. Rauws, P.L. Schuller, and G.J. Van Esch. 1976a. Toxicity of methylmercury chloride in rats I: short-term study. *Toxicology* 6:85-96.
- Verschuuren, H.G., R. Kroes, E.M. Den Tonkelaar, J.M. Berkvens, P.W. Helleman, A.G. Rauws, P.L. Schuller, and G.J. Van Esch. 1976b. Toxicity of methylmercury chloride in rats III: long-term toxicity study. *Toxicology* 6:107-123.
- WHO (World Health Organization). 1989. Mercury - environmental aspects. *Environmental Health Criteria* 86. Geneva.
- Wobeser, G., N.O. Nielsen, and B. Schiefer. 1976a. Mercury and mink I. The use of mercury contaminated fish as a food for ranch mink. *Can.J.Comp.Med.* 40:30-33.
- Wobeser, G., N.O. Nielsen, and B. Schiefer. 1976b. Mercury and mink II. Experimental methyl mercury intoxication. *Can.J.Comp.Med.* 40:34-45.

Ecotoxicological Profile for Ecological Receptors

Selenium

This ecotoxicological profile on selenium contains five subsections: (1) background (e.g., background concentrations), (2) geochemistry of the constituent in various ecological media, (3) effects characterization, (4) bioaccumulation potential, and (5) criterion development. The first four sections are intended to provide an overview of the environmental factors that influence the toxicological potential of selenium so that the limitations of the criteria may be better understood. The fifth section presents the rationale and development of criteria for the suite of ecological receptors used to represent the aquatic and terrestrial ecosystems. The profile is intended to present the ecotoxicological criteria in a broader environmental context so that the ecological significance of the criteria may be properly interpreted.

I. Background

Selenium is an essential nutrient for many ecological receptors, but it has also been implicated in deleterious effects at high concentrations. The range between beneficial and harmful levels is quite narrow such that concentrations that are required for some species may inhibit physiological processes in other species. For example, the recommended concentration of selenium for freshwater aquatic organisms is approximately 35 ppb; however, concentrations from 60 to 600 ppb result in mortality to sensitive aquatic organisms. Therefore, an increase in 25 ppb of selenium can result in adverse effects to some receptors. The biological response of organisms will vary depending on the species, age, tolerance, and the chemical form of selenium.

The environmental behavior of selenium is complex and not well characterized; however, it is an issue of current research. The bioaccumulation and biomagnification of selenium in aquatic and terrestrial receptors has been observed. Adverse impacts to receptors at high trophic levels (i.e., mammals and birds) has been well documented in case studies conducted at the Kesterson Reservoir, San Joaquin River, and Belews Lake, NC.

II. Geochemistry of Selenium in Various Ecological Media

General

Knowledge of selenium speciation and partitioning within various environmental compartments is important in the evaluation of potential risks arising from toxicity. Selenium can exist in a variety of oxidation states (-2, 0, +4, +6), in both organic and inorganic compounds. Since the different oxidation states of selenium are characterized by unique solubilities and affinities for solid phases, changes from one oxidation state to another affect the potential mobility in the environment. Hence, the wide variations in selenium solubility and sorption characteristics among its different forms require that its speciation be understood in order to predict transport between environmental compartments.

- # Selenium can exist in four oxidation states (-2, 0, +4, +6). In aqueous environments, selenium is limited to the -2, +4, and +6 oxidation states.
- # Selenium is biologically active and can form organic as well as inorganic compounds.
- # The specific oxidation state and chemical form largely determine selenium's behavior in the environment.

The geochemical behavior of selenium in the environment is strongly dependent upon its oxidation state and specific chemical species. Selenium occurs in four oxidation states: -2, 0, +4, and +6. Selenium⁶⁺ and Se⁴⁺ occur as the oxyanions selenate (SeO₄²⁻) and selenite (SeO₃²⁻ and HSeO₃⁻), respectively. Elemental Se (Se⁰) occurs in colloidal form; whereas, selenide (Se²⁻) occurs as a variety of organic and inorganic selenides, including volatile methylated forms.

The specific chemical species will depend to a large degree on its oxidation state, which, in turn, is influenced by pH and Eh. Thermodynamically, selenate (SeO₄²⁻) should be the stable selenium species in oxic and alkaline environments; however, data from natural systems indicate that speciation is complex and cannot be predicted based on thermodynamics alone. Specifically, thermodynamics do not take into account biological production of apparently unstable species, nor the apparent stabilities of thermodynamically predicted unstable species due to kinetic hindrances to equilibrium (Doyle et al., 1995).

Selenium in Soils

The amount of selenium in soils is determined primarily by natural geochemical processes such as the weathering of parent bedrock materials or volcanic exhalations; however, anthropogenic sources may also contribute selenium to the soil system. Anthropogenic sources include coal/oil combustion facilities, selenium refining factories, base metal smelting and refining factories, mining and milling operations, as well as fertilizer applications and incineration of tires, paper, and municipal waste (ATSDR, 1996).

Selenium speciation in soils is a function of soil pH and Eh. Selenium may occur in a number of different forms, including elemental selenium, selenides, selenites, selenates, and organic selenium. Elemental selenium (Se⁰) is formed by bacteria, fungi, and algae, which are capable of reducing selenites and selenates. Elemental selenium is moderately stable in soils and is essentially insoluble, thus representing an inert sink under anoxic conditions.

Heavy metal selenides and selenium sulfides are also largely insoluble. They predominate in acidic soils and soils characterized by high organic matter content. Heavy metal selenides and selenium sulfides are generally considered immobile in soil. This is due to the low solubility that characterizes metal selenides such as copper and cadmium.

Elemental selenium can be oxidized to form selenites and selenates. The selenites (SeO_3^{2-}) are stable, under moderately reducing conditions, in alkaline to mildly acidic environments (Shamberger, 1983; Tokunaga et al., 1997). Although the selenites are soluble, they can strongly sorb onto surfaces of common soil minerals (iron oxides) and organic matter (Tokunaga et al., 1997). Selenites may also be removed from pore waters through the formation of an insoluble precipitate (basic ferric selenite $[\text{Fe}_2(\text{OH})_4\text{SeO}_3]$), which can be formed in acidic soils ($4.5 < \text{pH} < 6.5$). Geering et al. (1968) indicated that the selenite concentration in solution in soils is governed primarily by this ferric oxide-selenite complex.

- ul style="list-style-type: none;">
- # Selenium speciation is a function of soil pH and Eh.
- # Depending upon pH and Eh, selenium may occur as elemental selenium, selenides, selenites, selenate, and organic selenium.
- # Elemental selenium occurs under anerobic conditions. It is relatively stable and insoluble.
- # Selenides predominate in acidic soils and soils with high organic content. They are also relatively stable and insoluble.
- # Selenites are thermodynamically stable under reducing conditions, but may exist under oxidizing conditions as well. They are stable in alkaline to mildly acidic environments. Although they are soluble, they sorb onto iron oxides and organic matter, thereby limiting their mobility in the environment.
- # Selenate is the predominant species at pH values greater then 6.5 and oxidizing conditions. It is characterized as being soluble and having a low sorption potential. It is readily available for uptake by plants.
- # A variety of organic complexes may exist. These complexes are most prevalent in high organic soils.

At pH values greater then 6.5, selenium may be oxidized to the more soluble selenate ions (SeO_4^{2-}). Because of its relatively high solubility and low tendency to sorb onto soil particles, selenates are readily available for transport and uptake by plants. Soluble selenate (principally sodium selenate) appears to be responsible for most of the naturally occurring accumulation of high selenium in plants.

Selenium in organic complexes occurs in varying quantities in soils. Organic species of selenium can be increased by the accumulation of decaying plant residues. Organic selenium is also subject to microbiological breakdown, resulting in alkylselenium compounds, mainly dimethylselenide. In humic temperate regions with the relatively greater accumulation of soil organic matter, organic-selenium forms assume more importance. Organic soils retain selenium more strongly than mineral soils. Studies have shown that the addition of organic matter greatly diminished the evolution of volatile selenium compounds as well as the movement and leaching of selenium through soil columns (Berrow and Ure, 1989).

Based on the behavior of selenium in soils, it is expected that selenium would be concentrated in soil horizons characterized by either high iron contents or high organic matter contents. In New Zealand soil profiles, Wells (1967) as cited in Berrow and Ure (1989) found

that B2 horizons, with their accumulation of iron and clay-sized colloids, were characterized by the greatest selenium concentrations. In another study conducted in the United States, selenium concentrations were found to range from 0.01 to 2.5 mg/kg in 11 soil profiles collected in the United States (Berrow and Ure, 1989). The most ferruginous horizons of the soils were found to be the most seleniferous. In acid ferruginous soils, selenium was bound as a basic ferric selenite or strongly absorbed on ferric oxide. Lateritic soils of the continental United States that have been analyzed also contain 0.5 to 2.4 mg/kg of selenium in the iron-rich horizons (Shamberger, 1983).

An accumulation of selenium in podzolic B horizons and organic surface horizons was found in 54 Canadian profiles by Levesque (1974). In Finnish soils, Koljonen (1975) found that selenium was enriched in the O-A1 horizons rich in organic matter and in the iron-rich B horizons. Multiple regression analyses revealed that the predominant factors involved in selenium distribution were the content of the parent material and the organic carbon content of the upper soil horizons (Berrow and Ure, 1989).

Selenium in Surface Water

The data for selenium in surface water can be divided into two operationally defined fractions: dissolved selenium (passes through filters with 0.45- μm openings), and particulate selenium (trapped by filters having $\geq 0.45\text{-}\mu\text{m}$ openings, typically suspended sediment and other suspended solids). Particulate selenium exists in the same oxidation states as dissolved selenium. Dissolved selenium exists in three oxidation states, including selenide (Se^{2-}), selenite (Se^{4+}), and selenate (Se^{6+}). Although not truly dissolved, colloidal selenium passes through filters having 0.45- μm openings and, as a consequence, is grouped with the dissolved selenium phase. Colloids may consist of elemental selenium (Se^0).

Note that thermodynamic calculations describing selenium geochemistry can be misleading. In fact, thermodynamically unstable species have been measured at significant concentrations in natural waters. The presence of these species is attributed to biological mediation and/or kinetic hindrances to equilibrium.

- # Selenate is the thermodynamically stable species under oxic and alkaline conditions.
- # Selenite may also exist and should be assumed to be present.
- # Elemental selenium and selenides dominate under anoxic conditions.
- # Organic selenides may be present under both oxic and anoxic conditions.

Although selenate is the thermodynamically stable species under oxic and alkaline water conditions, both selenite and selenate are common in surface waters (ATDSR, 1996). Selenite exists as HSeO_3^- at pH 6. As the pH increases, the concentration of HSeO_3^- becomes less prevalent and SeO_3^{2-} increases in importance. At a pH of 9, SeO_3^{2-} exceeds HSeO_3^- by a ratio of about 2:1. Dissolved selenate is present as SeO_4^{2-} in oxic waters having a pH range of 6 to 9.

The thermodynamic models predict that elemental selenium and selenide should dominate under anoxic conditions. Selenide may be present as H_2Se and HSe^- in anoxic waters. It

may also be present as organic selenides (primarily selenoamino acids bound in soluble peptides) in oxic and anoxic waters.

Although selenate is expected to be the dominant form of selenium in surface water, significant variability in speciation exists. In the Susquehanna River, which empties into the Chesapeake Bay, selenate is the predominant form of dissolved selenium (69% of the total). In contrast, samples from the St. Lawrence River in Canada show selenite to be the selenium species of highest concentration (67% to 76% of the total). Furthermore, recent data for several rivers in North America show that selenite and organic selenide (Se^{2-} and Se^0) are the dominant species. Specifically, it was found that 77% of the inorganic selenium can be classified as colloidal, whereas 70% of the organic selenium is colloidal, in river water collected from the James River in Virginia.

Because selenium is of special concern in the western United States due to widespread areas of selenium-rich source rocks, arid climate, and the potential for evapoconcentration, factors controlling transport and behavior in arid fluvial systems were investigated by Doyle et al. (1995). The three river systems included the Truckee, Walker, and Carson River watersheds, which comprise an area of over 200,000 km² in eastern California and western Nevada.

Selenium concentrations of < 1 to ~ 3 nM were measured in the three watershed systems. The source of the selenium appears to be atmospheric input and not geologic weathering. Despite the ability of selenium to evapoconcentrate, evidence indicated that it did not behave conservatively and was, in fact, depleted relative to other conservative species. Possible removal mechanisms include:

- # Selenate reduction in anoxic bottom sediments and/or waters of the terminal lakes
- # Volatilization to the atmosphere via planktonic biomethylation
- # Incorporation of selenium-rich organic matter into sediments and subsequent burial
- # Adsorption of thermodynamically unstable selenium onto iron oxides.

Selenium in Sediments

Transport across boundaries between surface waters and the underlying sediments is important in understanding the cycling of selenium. On a total mass basis, most of the selenium in surface water-sediment systems can be found in the sediments (Cutter, 1989). Selenium may be associated with the organic material, iron and manganese oxides, carbonates, or other mineral phases that constitute a sediment particle. This association is attributed to abiotic and biotic

- # Most of the selenium in surface water-sediment systems is found in the sedimentary phase.
- # Total dissolved selenium decreases more rapidly when organic matter is present in the system.
- # Accumulations of selenium within sediments are largely confined to the near surface.
- # Reducing conditions in the sediment promote the reduction of selenite and selenate to elemental selenium.

scavenging of dissolved ions from the water column and burial in the underlying sediments. Abiotic scavenging includes selenium adsorption and/or coprecipitation (primarily selenite and selenate). Selenide can be covalently bound in the organic portion of a sediment (the association of selenide with organic materials in sediment reflects the reducing conditions typical of organic matter). In addition, selenium may be found in anoxic sediments as insoluble metal selenide precipitates, as insoluble elemental selenium, or as ferroselite (FeSe_2) and selenium-containing pyrite.

In experiments designed to determine trends in inorganic selenium concentrations in water columns associated with sediment and sediment augmented with organic matter, it was found that there was a net decrease in total dissolved selenium in the water columns of both sediment systems (Tokunaga et al., 1997). More rapid decreases were observed in systems having organic matter added to the sediment. By the end of the experiment, 25% of the original selenium in the surface waters was transported into the unamended sediments. For systems, amended with organic matter, 95% of the selenium originally in the ponded water was transported into the sediments. Accumulations of selenium within the sediments were largely confined to the near-surface regions (< 25 mm depth) in both sets of experiments. Reducing conditions in the sediment promoted the reduction of selenate to selenite to elemental selenium, allowing a net accumulation of insoluble selenium species. The highest accumulations of selenium in the sediment occur within the top 1 mm of the columns, indicating a rapid reduction to elemental selenium.

Selenium concentrations in sediment are generally in the range of 1.5 to 4 mg/kg (Cutter, 1989). However, sedimentary accumulation of selenium will depend on a number of factors, including the total dissolved concentration of selenium in the system, sedimentation rate, biological productivity, and sediment type. Sediments in reservoirs that receive fossil fuel combustion products (e.g., fly ash) are characterized by elevated selenium concentrations. Cutter (1986) analyzed the concentration and phase distribution of selenium in sediments from three power plants-receiving waters (coal fly ash was the major source of selenium in the receiving waters). Within the sediments, selenium ranged in concentration from 6.5 to 29 mg/kg. Cutter (1986) indicated that more than 90 percent of the selenium was present in an "organic phase"; however, this organic phase is considered an operational definition and may include both elemental selenium and/or a selenium sulfide phases.

III. Effects Characterization

This section, along with the bioaccumulation potential section, is subdivided to evaluate receptors of the freshwater and terrestrial ecosystems separately. Figure J-3 summarizes the range of effects data for receptors of concern illustrating the sensitivity of various taxa to exposure. For reference, the water quality standards for freshwater communities (NAWQC or secondary values) are included for both acute and chronic endpoints. These values can be disregarded for receptors in the terrestrial community, because the NAWQC only provides protection for aquatic receptors, not predators of aquatic biota. The NAWQC provide a context for effects ranges in the aquatic community.

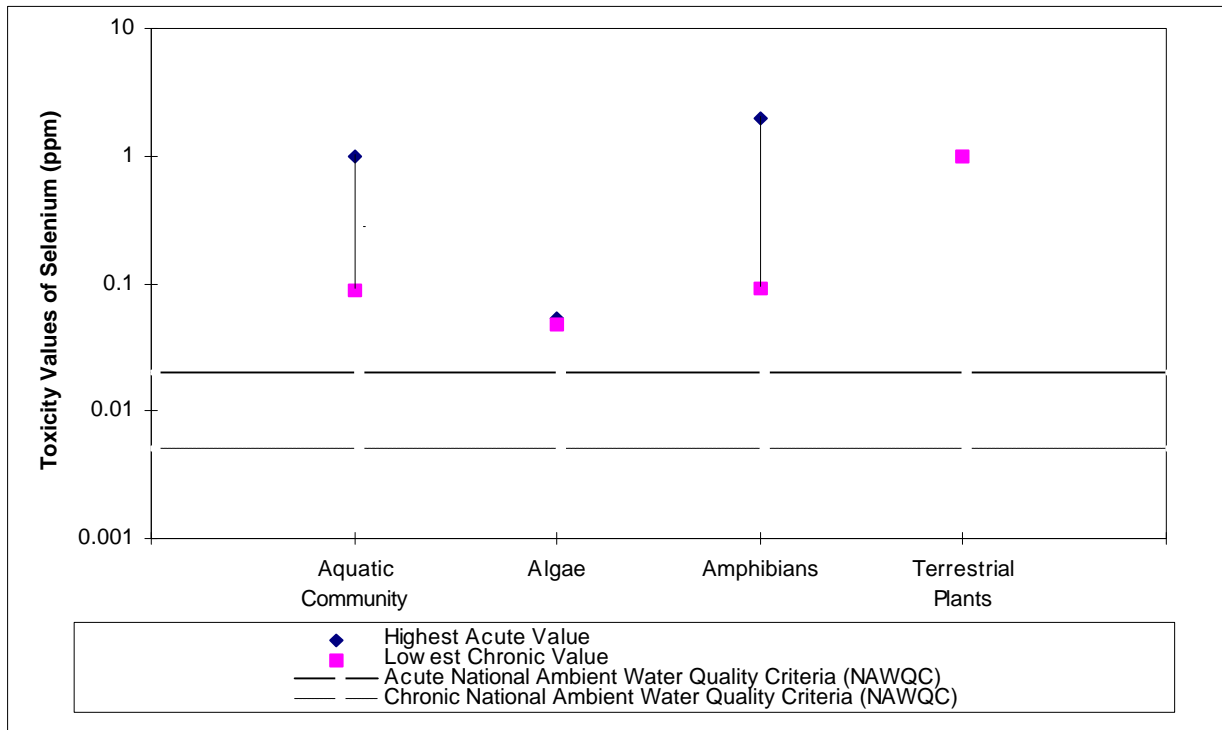


Figure J-3. Selenium: effects ranges for selected ecological parameters.

Freshwater Ecosystems

Sensitive aquatic organisms exhibit increased mortality at water concentrations of selenium between 60 and 600 ppb selenium (chemical form unspecified). Selenium's toxic effects in fish may vary with life stage, but include behavioral changes, altered blood chemistry, and decreased reproductive success (Eisler, 1985). Selenite is significantly more toxic than selenate, and younger life stages are more sensitive than older (Hamilton and Buhl, 1990). For selenite, LC_{50} values of 13.8 mg/L for chinook salmon and 7.8 mg/L for coho salmon were reported; for selenate, the corresponding values were 115 mg/L and 33 mg/L. Aquatic invertebrates demonstrate higher sensitivity to acute exposures than fish, with LC_{50} values ranging from 0.07 to 0.8 mg/L (Eisler, 1985). Amphibians exposed to water concentrations of selenium as sodium selenite have also shown adverse effects. Exposure to 2.0 mg/L and above during the egg stage of *Xenopus laevis* caused developmental malformations (chemical form unknown). Exposure during the tadpole stage resulted in altered behavior and physiological function (Power et al., 1989). Lethality to amphibians was observed in surface water concentrations ranging from 7 to 11 mg selenium/L (as sodium selenate). In algal communities, concentrations between 47 and 53 ppb have resulted in inhibited growth and shifts in representative species. No ecotoxicity data on potential effects to the sediment community could be identified (Power et al., 1989; Schuytema and Nebekar, 1996).

Terrestrial Ecosystems

Both acute and chronic effects have been indicated in terrestrial receptors. Acute selenosis in livestock may result from ingestion of highly contaminated plants and may produce death (Eisler, 1985). Plant materials containing 400 to 800 ppm selenium have been found to produce acutely toxic effects. The minimum orally administered lethal dose, in mg Se/kg body weight, range from 3.3 for horses, to 11 for cattle, to 15 for swine (Eisler, 1985).

Chronic selenosis in mammals may result from dietary exposures ranging from 1 ppm (rat) to 44 ppm (horse) and drinking water exposures of 0.5 to 2.0 ppm (Harr and Muth, 1972). Selenosis has also been associated with reproductive anomalies, including congenital malformations and growth retardation (Eisler, 1985). Rats dosed with selenium as selenate at 0.34 mg/kg-day for two generations demonstrated decreased reproductive success (Rosenfeld and Beath, 1954). In general, studies on rats, mice, swine, and cattle, have found that the young born to females with selenosis were emaciated, were unable to nurse, were part of small litters, and exhibited high mortality rates (Eisler, 1985). Although some studies have reported carcinogenicity, selenium's carcinogenic potential remains unclear (U.S. EPA, 1998).

Limited literature sources were identified to evaluate the direct impacts to terrestrial plant and soil communities; however, overall impacts appear less severe in these receptors. Their role as bioaccumulators and vectors of exposure to higher trophic levels may play a more significant part in the observed ecological impacts of selenium. In plants, the lowest observed effects concentrations have been reported in the range of 1 to 4 ppm; whereas, acutely toxic concentrations of 25 to 50 ppm have been observed (Efroymson et al., 1997b; Eisler, 1985). One study identifying reproductive effects of selenium to cocoon production in earthworms reported no effects at 77 ppm.

IV. Bioaccumulation Potential

Freshwater Ecosystems

Selenium accumulates in the aquatic environment in many kinds of organisms, including algae, periphyton, daphnids, benthic insects, annelids, molluscs, crustaceans, and fish, as well as birds (Besser et al., 1993; Lemly, 1985; Ohlendorf et al., 1990). Ohlendorf et al. (1990) studied accumulation of selenium in aquatic birds living near contaminated waterbodies. They found that selenium concentrations in liver tissues of birds from this site were much higher, often ten times or more, than those of birds living in relatively uncontaminated reference sites. Evidence suggests that accumulation of selenium occurs more readily as organoselenium compounds than as inorganic forms. Preferential uptake of selenomethionine relative to inorganic species has been reported in algae, daphnids, and fish (Lemly, 1985; Besser et al., 1993). Consumption of selenomethionine has also been shown to be more effective than sodium selenite in raising the selenium content of bird tissues and eggs (Eisler, 1985). Low concentrations of Se-methionine could thus contribute significantly to selenium bioaccumulation and toxicity in aquatic biota, although the chemical forms and concentrations of specific organoselenium compounds are not often reported in the literature, making assessments of their toxicological importance difficult (Besser et al., 1993).

Bioaccumulation factors (BAFs) for selenium used to determine food chain exposures are based on studies from Lemly (1985). This important field study is based on selenium concentration in fish inhabiting a river basin where selenium enters the reservoir as part of coal ash disposal. Lemly (1985) suggests that selenium not only can biomagnify through the food chain, but that the large amount of selenium accumulated in higher trophic piscivorous fish can shut down their reproductive systems and, in many cases, cause death. Because this is a field study where the fish receive exposures of selenium via food and water, values presented in Lemly (1985) are BAFs. A muscle-based BAF of 1,692 L/kg is used to represent trophic level 4 fish for estimating food chain exposures to piscivorous mammals and birds; this value is based on the geometric mean of the BAFs 1,571, 2,019, and 1,527 L/kg from piscivorous fishes such as crappie (*Pomoxis* sp.), Largemouth bass (*Micropterus salmoides*), and white perch (*Morone americana*), respectively. Additionally, a BAF of 485 L/kg from blueback herring (*Alosa aestivalis*) and threadfin shad (*Dorosoma petenense*) represents BAFs for trophic level 3 fish for estimating food chain exposures to piscivorous wildlife. Because no whole-body BAFs are identified, the muscle-based BAFs are used. As an aside, all BAF values are taken from Table 4 of Lemly (1985); although they are presented in units of L/g, they seem to be too high for even the most bioaccumulative constituents. A closer examination on the concentration of selenium in fishes (Figure 4 and 5) and concentration of selenium strongly suggest that the values in Table 4 are in units of L/kg rather than in L/g.

Terrestrial Ecosystems

Bioaccumulation in terrestrial invertebrates, plants, and small mammals is currently being investigated at Oak Ridge National Labs. Bioaccumulation and bioconcentration factors for terrestrial plants, invertebrates, and small mammals have been proposed from review of primary literature sources. The 90th percentile of the bioaccumulation data for these receptors derived from both laboratory and field studies was used to determine terrestrial food chain exposures. For earthworms, a BAF of 1.3 was proposed for selenium based on 15 data points. For terrestrial plants, a BCF of 26 was proposed based on 237 data points. For small mammals, based on 35 reported values assessing the transfer of selenium from soil to small mammals, a BAF of 1.2 was proposed (Sample et al., 1997, 1998a, 1998b). These values are in the process of being reviewed for use in modeling food chain exposures to terrestrial species, but currently they stand as the most comprehensive collection of bioaccumulation data for terrestrial ecological receptors. Further review of methods and primary literature is currently being conducted on these high-end values (Sample et al., 1997, 1998a, 1998b).

V. Criteria Development

The benchmark values presented in this section for mammals and birds were used to derive protective media-specific criteria as outlined in the stressor-response profile methodology (i.e., analysis phase of ERA). By scaling the benchmark study by body weight to a representative wildlife receptor (e.g., rat study extrapolated to a shrew), determining the dietary preferences of wildlife receptor and the potential bioconcentration in prey, a protective concentration (i.e., criteria) in soil, plants, or surface water was developed. Since criteria for receptors other than mammals and birds were already in media concentrations, this same derivation process was not required. A summary of criteria is provided in Table J-3. Although criteria were developed for numerous wildlife receptors of both the aquatic (e.g., otter, mink, and great blue heron) and

terrestrial ecosystems (e.g. shrew, fox, and hawk), only the lowest criterion is presented in Table J-3. It is assumed that, by protecting the more sensitive species, the other receptors are protected as well.

Mammals: Rosenfeld and Beath (1954) examined the effects of selenium on the reproduction of successive generations of Wistar rats. The authors administered doses of 1.5, 2.5, and 7.5 ppm of selenium as selenate in drinking water. The 2.5-ppm dose was reported to have reduced the number of young reared by the second generation mothers by 50%. This reduction resulted in a LOAEL of 2.5 ppm and a NOAEL of 1.5 ppm. These effects levels correspond to daily doses of 0.34 and 0.20 mg/kg-day, based on the Wistar rat's reference body weight of 0.320 kg and water consumption rate of 0.043 L/day (U.S. EPA, 1988).

The NOAEL of 0.20 mg/kg-day for reproductive effects from the Rosenfeld and Beath (1954) study was chosen to derive the toxicological benchmark for the following reasons: (1) doses were administered over a chronic duration and via oral ingestion, an ecologically significant exposure pathway; (2) it focused on long-term reproductive success as a critical endpoint; (3) it contained dose response information; and (4) it resulted in the lowest toxicity value for a critical endpoint.

Schroeder and Mitchener (1971) assessed the reproductive effects of selenium in three generations of mice. A single dose of 3 ppm selenium as selenate was administered in drinking water. Mice in all three generations produced fewer offspring and a greater percentage of runts than the controls. Conversion of the 3-ppm dose to a daily dose in units of mg/kg-day required the use of an allometric equation for water consumption by laboratory mammals (U.S. EPA, 1988):

$$\text{Water Consumption (L/day)} = 0.10(W^{0.7377})$$

where W is body weight in kilograms. Using a reference body weight for two typical types of laboratory mice (0.035 kg) (U.S. EPA, 1988) and a calculated water consumption rate of 0.008 L/day, a daily dose of 0.69 mg/kg-day was calculated. Nobunaga et al. (1979) exposed mice to two oral doses of selenium as selenite in drinking water for 30 days prior to mating and for the first 18 days of gestation. No significant effects on reproduction or incidences of fetotoxicity were evident at the lower dose of 11.4 nmol/mL (NOAEL), however, the higher dose of 22.8 nmol/mL (LOAEL) resulted in a significant reduction in fetal growth. These effects levels correspond to daily doses of 0.9 mg/kg-day and 1.7 mg/kg-day. To arrive at these figures, the molecular weight of sodium selenite was used to convert the nmol/mL doses to ppm doses. The ppm dose was then converted to the daily dose by using the geometric mean of mice body weights (0.028 kg) given in the study, and a water intake rate of 0.007 L/day, calculated from the allometric equation presented above (U.S. EPA, 1988).

The Schroeder and Mitchener (1971b) study was not chosen for the derivation of the benchmark because it did not contain sufficient dose response information. The Nobunaga (1979) study was not chosen because it did not report the lowest toxicity value for a critical endpoint.

Table J-3. Selenium Criterion in Soil, Sediment, Surface Water, and Plant Tissue Developed for Each Representative Receptor

Receptor	Criteria	Units	Exposure Pathway	Representative Species	Reference
Aquatic					
Mammals	6.0E-03	mg/L water	Food web	River Otter	Ambrose et al., 1976
Birds	1.9E-02	mg/L water	Food web	Kingfisher	Heinz et al., 1987
Algae and Aquatic Plants	1.0E-01	mg/L water	Direct contact	<i>Scenedesmus obliquus</i>	Suter and Tsao, 1996
Freshwater Community					
Total	5.0E-03	mg/L water	Direct contact	Aquatic biota	U.S. EPA, 1995
Selenium IV	2.8E-02	mg/L water	Direct contact	Aquatic biota	U.S. EPA, 1995
Selenium VI	9.5E-03	mg/L water	Direct contact	Aquatic biota	U.S. EPA, 1995
Amphibian (acute effects)	1.6E+00	mg/L water	Direct contact	Various amphibian species	Power et al., 1989; Schuytema and Nebeker, 1996
Terrestrial					
Mammals	2.1E+01	mg/kg soil	Food web	Raccoon	Rosenfeld and Beath, 1954
Birds	1.1E+01	mg/kg soil	Food web	American woodcock	Heinz et al., 1987
Mammals	1.1E+00	mg/kg plant tissue	Food web	Meadow vole	Rosenfeld and Beath, 1954
Birds	1.1E+01	mg/kg plant tissue	Food web	Northern bobwhite	Heinz et al., 1987
Plant Community	1.0E+00	mg/kg soil	Direct contact	Sorghass	Efroymsen et al., 1997b
Soil Community	7.0E+01	mg/kg soil	Direct contact	Soil invertebrates	Efroymsen et al., 1997a

The same surrogate species study (Rosenfeld and Beath, 1954) was chosen to derive the selenium benchmark for mammalian species representing the terrestrial ecosystem.

Birds: Only one study was identified that investigated the effects of selenium toxicity on avian species. Mallard duck pairs were fed diets containing selenium for 4 weeks prior to egg laying at doses of 1, 5, 10, 25, and 100 ppm selenium as sodium selenite (Heinz et al., 1987). There were no effects on the weight or survival of adults at the 1-, 5-, and 10-ppm dose levels. At the 25-ppm level females took longer to begin laying eggs and intervals between eggs were longer. Survival of ducklings in the 25-ppm group was lower than in the lower exposure groups. Among ducks fed 10 ppm and 25 ppm, there was a significantly greater frequency of lethally deformed embryos, as compared to the lower exposure treatment groups. This resulted in a LOAEL of 10 ppm and a NOAEL of 5 ppm. These effects levels correspond to daily doses of 1.0 and 0.5 mg/kg-day, respectively, converted by using the food intake rate of 105.5 g/day and the geometric mean (1.055 kg) of the control body weights given in the study.

The NOAEL of 0.5 mg/kg-day from the Heinz et al. (1987) study was selected to derive the avian benchmark value for the freshwater ecosystem because: (1) chronic exposures were administered via oral ingestion; (2) reproductive toxicity was one of the primary endpoints examined, and (3) the study contained sufficient dose-response information.

As in the freshwater ecosystem, the study by Heinz et al. (1987) was used to calculate the benchmarks for birds in the generic terrestrial ecosystem.

Freshwater Community: Two sources were evaluated in selecting criteria for the protection of aquatic biota: (1) Final Chronic Values (FCV) derived under the Great Lakes Water Quality Initiative (GLWQI) (U.S. EPA, 1995) and (2) National Ambient Water Quality Criteria (NAWQC) published by the EPA Office of Water. The FCVs of 5.0E-03 mg/L for total selenium, 2.8E-02 mg/L for selenium IV, and 9.5E-03 mg/L for selenium VI developed under the GLWQI were selected as the appropriate criteria to use in this analysis. The GLWQI values were considered preferable to the NAWQC because: (1) the GLWQI values are based on the same methodology used to develop NAWQC (i.e., Stephan et al., 1985); (2) the NAWQC data set was augmented with previously unavailable acute and chronic toxicity data; and (3) species taxa used to generate the GLWQI values are suitable for national application since they include species and taxa found throughout the United States.

Although total concentrations of metals are still deemed scientifically defensible by EPA, recent EPA guidance recommends the use of dissolved metals concentrations to better reflect the bioavailability of metals (e.g., Prothro, 1993). EPA has developed conversion factors (CFs) to estimate probable dissolved concentrations of metals in surface waters given a total metal concentration as described in 60 FR22231 (*Water Quality Standards...Revision of Metals Criteria*). A CF is not yet available for selenium. This adjustment reflects the current EPA position on criteria development and regulatory application of metals; however, the issue of metal bioavailability in surface waters is the topic of intensive research (e.g., Bergman and Dorward-King, 1997). The final surface water criterion for selenium species is presented in Table J-3.

Amphibians: No suitable subchronic or chronic studies were identified that studied the effects of selenium toxicity on reproductive or developmental endpoints in amphibian species. Acute toxicity data on selenium were identified to range from 7 to 11 mg/L during embryo exposures of *Xenopus laevis*. Low effects and no effects data were identified in one study with reported values of 1.6 and 0.8 mg selenium/L, respectively (Schuytema and Nebeker, 1996). Using this range as a guide, both of these values fall above the NAWQC; however, lacking sufficient data on various species, exposure durations, and life stages, the assertion of protection under the NAWQC cannot be made. Incorporating the amphibian data into the NAWQC within the data requirement categories is currently under consideration. Since amphibian species are more likely to breed in standing waters such as wetlands or ponds, the appropriateness of combining protection of amphibian receptors with the aquatic community is unclear (Power et al. 1989; Schuytema and Nebeker, 1996).

Algae and Aquatic plants: The benchmarks for aquatic plants were either: (1) a no observed effects concentration (NOEC) or a lowest observed effects concentration (LOEC) for vascular aquatic plants (e.g., duckweed) or (2) an effective concentration (EC_{xx}) for a species of freshwater algae, frequently a species of green algae (e.g., *Selenastrum capricornutum*). The benchmark value for selenium reported by Suter and Tsao (1996) was 1.0E+02 µg/L (selenate) based on the growth inhibition of the green alga *Scenedesmus obliquus* in 14-day chronic toxicity tests. The selection of a benchmark based on selenium as selenate is preferred because plants show preferential uptake of this form. Low confidence is placed in this criterion since it is only based on one study.

Benthic Community: The premier source of field sediment data is NOAA, which annually collects and analyzes sediment samples from sites located in coastal marine and estuarine environments throughout the United States as part of the National Status and Trends Program (NSTP). From the range of adverse effects data, criteria are developed estimating the 10th percentile effects concentration (ER-L) and a median effects concentration (ER-M) for adverse effects in the sediment community (Long et al., 1995). A second criteria document evaluated for sediment criteria development was the *Approach to the Assessment of Sediment Quality in Florida Coastal Waters Volume 1- Development and Evaluation of Sediment Quality Assessment Guidelines* (MacDonald et al., 1994) published by the Florida Department of Environmental Protection (FDEP). The criteria developed by FDEP were also based on the NOAA data; however, the method of derivation of the criteria was changed. Neither of these documents developed a suitable sediment benchmark for selenium. Therefore, no benchmark on selenium could be developed.

Terrestrial Plants: As presented in Efroymson et al. (1997b), phytotoxicity benchmarks were selected by rank-ordering the LOEC values and then approximating the 10th percentile. If fewer than 10 studies were available, the lowest LOEC was selected as the benchmark. Such LOECs applied to reductions in plant growth, yield, or seed elongation, or other effects reasonably assumed to impair the ability of a plant population to sustain itself. The selected benchmark for phytotoxic effects of selenium in soils is 1.0 mg/kg (Efroymson et al., 1997b). The derivation of the criterion is based on 13 phytotoxicity data points on various agricultural (e.g., barley, ryegrass) species measuring growth endpoints such as height and weight of shoots and roots. Considering this criterion was based on multiple studies over a range of species, confidence in this benchmark is high.

Soil Community: Because no adequate data to develop community-based criteria were identified, criteria for soil from earthworm studies presented in Efroymson et al. (1997a) of 70 mg/kg for selenium was used; it is based on one study reporting effects on growth and reproduction of *Eisenia fetida*. Earthworms have been recognized to play important roles in promoting soil fertility, releasing nutrients, providing aeration and aggregation of soil, as well as being an important food source for higher trophic level organisms. Even though earthworms are important, basing a soil criteria on one species does not ensure protection to the entire soil community given the complex processes and interactions characteristic of functional soil communities.

References

- Ambrose, A.M., P.S. Larson, J.F. Borzelleca, and G.R. Hennigar, Jr. 1976. Long term toxicologic assessment of nickel in rats and dogs. *Journal of Food Science and Technology* 13:181-187. As cited in U.S. EPA 1998 (IRIS).
- ATSDR (Agency for Toxic Substances and Disease Registry). 1996. *Toxicological Profile for Selenium* (Update). Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA.
- Bergman, H.L., and E.J. Dorward-King (eds). 1997. *Reassessment of Metals Criteria for Aquatic Life Protection: Priorities for Research and Implementation*. SETAC Press, Pensacola, FL.
- Berrow, M.L., and A.M. Ure. 1989. Chapter 9: Geological materials and soils. In: *Occurrence and Distribution of Selenium*, M. Ihnat (ed.), CRC Press, Boca Raton, FL. pp. 213 to 242.
- Besser, J.M., T.J. Canfield, and T.W. La Point. 1993. Bioaccumulation of organic and inorganic selenium in a laboratory food chain. *Environmental Toxicology and Chemistry* 12:57-72.
- Cutter, G.A. 1986. *Speciation of Selenium and Arsenic in Natural Waters and Sediment. Volume I: Selenium Speciation*. EA-4641. Prepared for Electric Power Research Institute. Prepared by Old Dominion University, Norfolk, VA. pp. 5-1 to 5-12.
- Cutter, G.A. 1989. The estuarine behavior of selenium in San Francisco Bay. *Estuarine, Coastal, and Shelf Science* 28:13-34.
- Doyle, G.A., W.B. Lyons, G.C. Miller, and S.G. Donaldson. 1995. Oxyanion concentrations in eastern Sierra Nevada rivers - 1. selenium. *Applied Geochemistry* 10:553-564.
- Efroymson, R.A., M.E. Will, and G.W. Suter, II. 1997a. *Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates and Heterotrophic Processes: 1997 Revision*. ES/ER/TM-126/R2. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Lockheed Martin Energy Systems, Inc., Oak Ridge, TN.

- Efroymson, R.A., M.E. Will, G.W. Suter, II, and A.C. Wooten. 1997b. *Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision*. ES/ER/TM-85/R3. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Lockheed Martin Energy Systems, Inc., Oak Ridge, TN.
- Eisler, R. 1985. Selenium hazards to fish, wildlife, and invertebrates: a synoptic review. In: *Contaminant Hazard Reviews, Report No. 5*. U.S. Fish and Wildlife Service, U.S. Department of the Interior, Laurel, MD.
- Geering, H.R., E.E. Cary, L.H.P. Jones, and W.H. Allaway. 1968. Solubility and redox criteria for the possible forms of selenium in soils. *Soil Science Society of America Proceedings* 32:35-40.
- Hamilton, S.J., and K.J. Buhl. 1990. Acute toxicity of boron, molybdenum, and selenium to fry of chinook salmon and coho salmon. *Archives of Environmental Contamination and Toxicology* 19:366-373.
- Harr, J.R., and O.H. Muth. 1972. Selenium poisoning in domestic animals and its relationship to man. *Clinical Toxicology* 5(2):175-186.
- Heinz, G.H., D.J. Hoffman, A.J. Krynitsky, and D.M.G. Weller. 1987. Reproduction in mallards fed selenium. *Environmental Toxicology and Chemistry* 6:423-433.
- Koljonen, T. 1975. The behavior of selenium in Finnish soils. *Annales Agriculturae Fenniae* 14:240-247.
- Lemly, A.D. 1985. Toxicology of selenium in a freshwater reservoir: implications for environmental hazard evaluation and safety. *Ecotoxicology and Environmental Safety* 10:314-338.
- Lévesque, M. 1974. Selenium distribution in Canadian soil profiles. *Canadian Journal of Soil Science* 54:63-68.
- Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environmental Management* 19(1):81-97.
- MacDonald, D.D. 1994. *Approach to the Assessment of Sediment Quality in Florida Coastal Waters. Volumes I & II*. Prepared for Florida Department of Environmental Protection, Office of Water Policy. Prepared by MacDonald Environmental Sciences Ltd., Ladysmith, British Columbia.
- Nobunaga, T., H. Satoh, and T. Suzuki. 1979. Effects of sodium selenite on methylmercury embryotoxicity and teratogenicity in mice. *Toxicology and Applied Pharmacology* 47:79-88.

- Ohlendorf, H.M., R.L. Hothem, C.M. Bunck, and K.C. Marois. 1990. Bioaccumulation of selenium in birds at Kesterson Reservoir, California. *Archives of Environmental Contamination and Toxicology* 19:495-507.
- Power, T., K.L. Clark, A. Harfenist, and D.B. Peakall. 1989. A review and evaluation of the amphibian toxicological literature. In: *Technical Report Series No. 61*. Canadian Wildlife Service.
- Prothro, M.G. 1993. Office of Water Policy and Technical Guidance on Interpretation and Implementation of Aquatic Life Metals Criteria. Memorandum. U.S. Environmental Protection Agency,
- Rosenfeld, I., and O.A. Beath. 1954. Effect of selenium on reproduction in rats. *Proceedings of the Society for Experimental Biology and Medicine* 87:295-297.
- Sample, B.E., M.S. Aplin, R.A. Efroymsen, G.W. Suter, II, and C.J.E. Welsh. 1997. *Methods and Tools for Estimation of the Exposure of Terrestrial Wildlife to Contaminants*. ORNL/TM-13391. Prepared for Office of Environmental Policy and Assistance, U.S. Department of Energy. Prepared by Oak Ridge National Laboratory, Oak Ridge, TN.
- Sample, B.E., J.J. Beauchamp, R.A. Efroymsen, and G.W. Suter, II. 1998a. *Development and Validation of Bioaccumulation Models for Small Mammals*. ES/ER/TM-219. Prepared for Office of Environmental Management, U.S. Department of Energy. Oak Ridge National Laboratory, Oak Ridge, TN.
- Sample, B.E., J.J. Beauchamp, R.A. Efroymsen, G.W. Suter, II, and T.L. Ashwood. 1998b. *Development and Validation of Bioaccumulation Models for Earthworms*. ES/ER/TM-220. Prepared for Office of Environmental Management, U.S. Department of Energy. Oak Ridge National Laboratory, Oak Ridge, TN.
- Schroeder, H.A., and M. Mitchener. 1971. Toxic effects of trace elements on the reproduction of mice and rats. *Archives of Environmental Health* 23:102-106.
- Schuytema, G.S., and A.V. Nebeker. 1996. *Amphibian Toxicity Data for Water Quality Criteria Chemicals*. EPA/600/R-96/124. National Health and Environmental Effects Research Laboratory, Western Ecology Division, U.S. Environmental Protection Agency, Corvallis, OR.
- Shamberger, R.J. 1983. Biochemistry of selenium. In: *Biochemistry of the Elements*. Frieden, E. (ed.). 167-183. Plenum Press, New York, NY.
- Stephan, C.E., D.I. Mount, D.J. Hansen, J.H. Gentile, G.A. Chapman, and W.A. Brungs. 1985. *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses*. PB85-227049. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Duluth, MN.

- Suter, G.W., II, and C.L. Tsao. 1996. *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1996 Revision*. ES/ER/TM-96/R2. Prepared for Office of Environmental Management, U.S. Department of Energy. Prepared by Health Sciences Research Division, Oak Ridge National Laboratory Risk Assessment Program, Oak Ridge, TN.
- Tokunaga, T.K., G.E. Brown, Jr., I.J. Pickering, S.R. Sutton, and S. Bajt. 1997. Selenium redox reactions and transport between ponded waters and sediments. *Environmental Science and Technology* 31:1419-1425.
- U.S. EPA (Environmental Protection Agency). 1988. *Recommendations for and Documentation of Biological Values for Use in Risk Assessment*. EPA/600/6-87/008. Office of Health and Environmental Assessment, Cincinnati, OH.
- U.S. EPA (Environmental Protection Agency). 1995. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Aquatic Life in Ambient Water*. EPA-820-B-95-004. Office of Water, Washington, DC.
- U.S. EPA (Environmental Protection Agency). 1997. *Protocol for Screening Level Ecological Risk Assessment at Hazardous Waste Combustion Facilities. Volumes I and II* (Internal Review Draft). EPA-R6-096-003. Office of Solid Waste, Dallas, TX.
- U.S. EPA (Environmental Protection Agency). 1998. Integrated Risk Information System (IRIS) Database. Cincinnati, OH. August.